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THE
LARYNGOSCOPE.

VOL. LXI SEPTEMBER, 1951. No. 9

NORTH AMERICAN BLASTOMYCOSIS. A REVIEW OF THE LITERATURE AND A REPORT OF TWO CASES PRIMARY IN THE LARYNX.*

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This paper endeavors to point out the clinical and pathological similarities between primary laryngeal blastomycosis and laryngeal cancer. It presents a summary and brief analysis of the general literature concerning systemic blastomycosis, a more detailed investigation of those reports dealing with primary laryngeal blastomycosis, and a report of two cases, in each of which a mistaken diagnosis was made.

DEFINITION.

This discussion is limited to the North American form of olastomycosis (Gilchrist and Stokes, 1898) and avoids consideration of the European form, caused by a fungus of the genus cryptococcus, and of the South American form, which is caused by blastomyces brasiliensis.

HISTORICAL.

Gilchrist and Stokes[1] first described a case of cutaneous blastomycosis in 1894. They named the causative organism

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blastomyces dermatitidis, and were able to demonstrate it in stained tissue sections. Walker and Montgomery[2] described the first case of systemic blastomycosis in 1902. Since then there have been reports of approximately 380 cases, 73 of which have been of the systemic variety.[3] In 1914, Stober[4] and his co-observers in the Chicago area published a monumental work concerning the cases seen there.

In 1918, Dennis[5] made the first report of primary laryngeal blastomycosis. In 1926, Jackson[6] reported a single case with laryngeal and pulmonary infection, and New[7] listed two primary laryngeal cases in 1928. In 1936, in an article titled "Laryngeal Blastomycosis Complicating Carcinoma," Fuller[8] reported a case which, in the light of the conclusions of the present paper, is of particular interest.

OCCURRENCE.

North American blastomycosis is found widely distributed throughout the United States and Canada. For many years the disease seemed centered around Chicago, and was at one time referred to as "Chicago disease," since 50 per cent of all the reported cases resided in that city or in its environs. It has recently been recognized, with increasing frequency, in the southeastern states. Smith[9] reported 59 patients with blastomycosis in 250,000 admissions to the Duke Clinic in 15 years. He explains this unusual frequency of occurrence by stating that "the southeastern states constitute an endemic area for this disease." It seems possible, however, that cases of blastomycosis sometimes occur more frequently in other areas than is recognized. Wade and Bel,[10] for instance, discovered five cases over a period of two years in New Orleans, where none had previously been recognized.

Though no age is immune, 50 per cent of all cases occur in individuals over 40 years of age, and males are affected nine times more frequently than are females.

Dennis[11] and Stober[12] stress the effect of occupation and living conditions on the onset and progress of the disease.

Damp, dusty, moldy rooms and generally poor living conditions seem to predispose to blastomycosis. There is no evidence of transfer by contagion.

MYCOLOGY.

The technique of microscopic examination for fungus with slide preparations of potassium hydroxide and scrapings, pus, sputum, or other suspected material, is fully described in the "Manual of Clinical Mycology,"[13] from which the authors and the W. B. Saunders Co. have permitted me to reproduce three

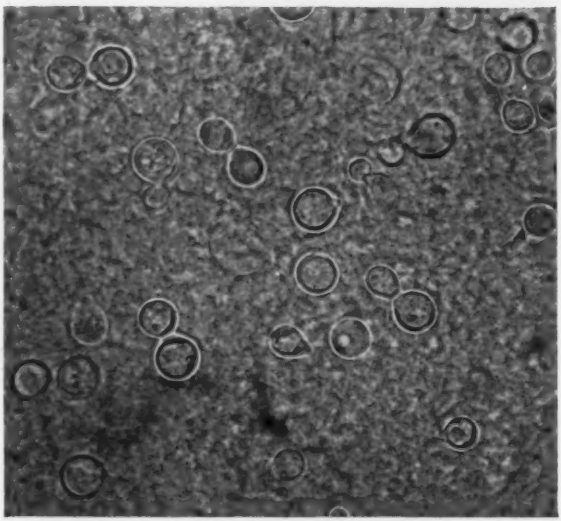

Fig. 1. Round, double-contoured, budding yeast-like cells in pus from subcutaneous abscess. × 700.

plates illustrating: *1.* blastomycosis dermatitidis as it appears in these microscopic preparations, *2.* the growth obtained on beef glucose agar at 37° C. for 23 days, and *3.* a colony on Sabouraud's glucose agar at room temperature for 23 days (see Figs. 1, 2 and 3).

PATHOLOGY.

Pathology textbooks devote little space to blastomycosis, and some fail even to mention epithelial hypertrophy as a prominent feature. This last omission is perhaps accounted for by the fact that much of their material for study is obtained at postmortem examinations, or from generous biopsy specimens which permit a study of the disease process of the organ as a whole. Eggston and Wolf,[14], writing a specialized textbook of ear, nose and throat pathology, point out the possibility of

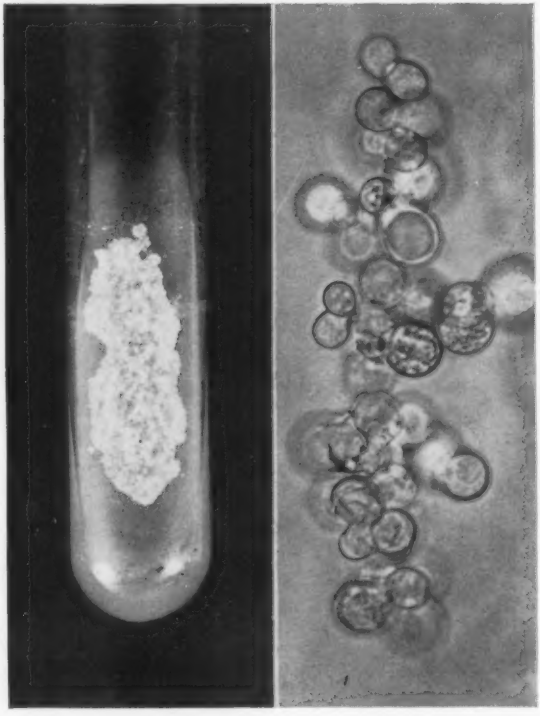

Fig. 2. (a) Yeast-like culture, 21 days, on beef infusion glucose agar at 37° C. (b) Budding yeast-like cells from beef infusion glucose agar at 37° C. × 700.

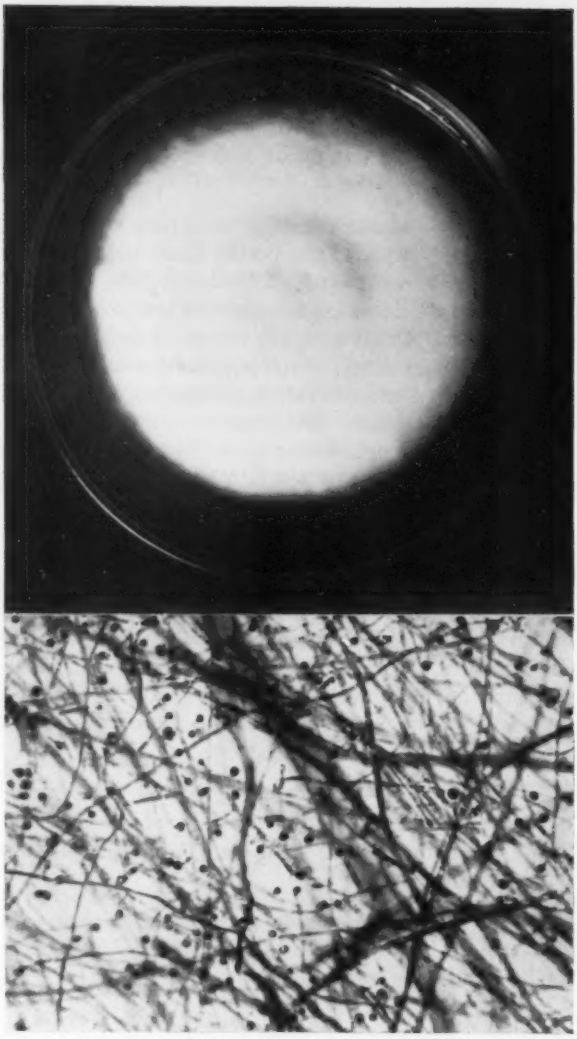

Fig. 3 (a) Filamentous colony on Sabouraud's glucose agar, 23 days, at room temperature. (b) Round and pyriform conidia found in filamentous stage from Sabouraud's glucose agar at room temperature. × 700.

confusion with carcinoma. Constant experience with the difficulties inherent in the interpretation of specimens which are small and often far from all inclusive, such as those obtained from biopsy of the larynx, may add to one's awareness of the dangers of diagnostic misinterpretation. There is, of course, great variance in the extent of the epithelial hypertrophy itself.

Undoubtedly the tissue reaction to injury by blastomyces dermatitidis varies widely, depending upon the host's immunologic reactions and the sites of invasion. The most characteristic response, as noted by most observers, is multiple small abscess formation. Giant cells are common, and the organism may be found within them; in some areas the organism cannot be demonstrated. Caseous necrosis, tubercle formation, and fibrosis may be prominent. The discovery of spherical, double-contoured, budding, thick-walled organisms eight to 15 micra in diameter is considered by some to be diagnostic.

IMMUNOLOGY.

Hypersensitivity to the antigen of blastomyces may be demonstrated by a positive skin test to blastomyces vaccine, and humoral antibodies can be detected by a complement fixation test. D. T. Smith,[15] who feels that the prognosis in blastomycosis depends on the immunologic state, studied 40 cases of blastomycosis at the Duke Clinic, and found the following immunologic groups:

1. *Skin test positive—complement fixation negative.* Prognosis in this group is relatively good, since these patients respond well to treatment after desensitization with vaccine.
2. *Skin test positive—complement fixation positive.* The response to treatment in this group is not so satisfactory, but may be good following desensitization.
3. *Skin test negative — complement fixation positive.* The prognosis in this group is very poor, as this represents an anergic reaction.

4. *Skin test negative—complement fixation negative.* These are usually early cases in which hypersensitivity and complement-fixing antibodies have not yet developed. Obviously, this group of patients will ultimately reclassify themselves into one of the other three groups.

DIFFERENTIAL DIAGNOSIS.

The usual portal of entry of systemic blastomycosis is the lungs—Wade and Bel[16] state that 96 per cent of all autopsied cases having blastomycosis had lung involvement—and the lesions greatly resemble tuberculosis. The onset is usually ascribed to "taking a cold." Dyspnea, pain in the chest, cough, and expectoration of purulent or bloody sputum are frequent complaints. The duration may be from a few months to several years. Anyone suspecting blastomycosis would, therefore, expect to find lung involvement; however, such is not always the case, and one must be on the alert to recognize the disease when no lung involvement occurs.

The similarity to tuberculosis is noted even in localized sections, such as those involving the larynx. Other diseases to be differentiated are lues, Boeck's sarcoid, other fungus infections, and, as found in my own two cases, cancer. In these two cases, as will be subsequently noted, both the clinical and pathological picture greatly resembled carcinoma.

PROGNOSIS.

All observers have noted a marked difference between the two main forms of blastomycosis, so far as the prognosis is concerned. The common cutaneous type rarely causes death, and rarely spreads to become the systemic variety. The systemic variety is usually fatal. Martin and Smith[17] noted a 92 per cent mortality rate in such cases when they had been followed two years or longer.

TREATMENT.

It is obvious from the high mortality rate in systemic blastomycosis that the treatment of this form of the disease is

unsatisfactory. It has proven resistant to all antibiotic and chemotherapeutic agents to date. Skin testing with vaccine, and determination of the presence or absence of complement-fixing antibodies, as suggested by Smith, should be carried out before any treatment is instituted. All patients showing hypersensitivity, as manifested by a positive skin test to the vaccine, should first be desensitized. Potassium iodide may then be given by mouth, but, should any untoward symptoms appear, it should be immediately withheld, and vaccine therapy must be instituted. The iodide may then be readministered after a sufficient interval. Should signs of iodism appear, the potassium iodide must be withheld for a reasonable interval before it may be readministered in small doses.

X-ray therapy has proven a valuable adjunct, especially in the treatment of skin lesions. Surgical drainage, or surgical removal of localized, resectable lesions has been strongly advised by Wade and Bel.[18] Hamblen[19] *et al.* and Smith[20] have reported instances in which surgical excision has been an adjunct in part of the successful treatment of systemic blastomycosis.

REVIEW OF PREVIOUSLY REPORTED CASES OF NORTH AMERICAN BLASTOMYCOSIS WHICH PRIMARILY INVOLVED THE LARYNX.

Since only five instances of primary laryngeal blastomycosis have been reported in the literature, a brief resumé of each case, as described by the author, is given.

Case 1: The first reported instance of primary laryngeal blastomycosis was given by Dr. Frank L. Dennis, in 1918. The patient, a 39-year-old white male, dated the onset of his illness from a severe attack of tonsillitis contracted while working in a dusty grain elevator. His condition improved temporarily, but became worse and settled in his larynx when he was sent to clean out the "dump," or pit, of the elevator, which contained decayed wheat, rotten wood, and rat manure. The patient was admitted to a sanitorium in 1916. Examination, however, failed to reveal pulmonary involvement. The laryngologist described "an infiltrated and ulcerated area involving the epiglottis and median glossoepiglottic ligament, both arytenoid regions, and the false cords." The biopsy showed blastomycosis.

Weekly X-ray treatments were given over the laryngeal area, but the patient was unable to tolerate adequate doses of potassium iodide. A tracheotomy became necessary, and pulmonary involvement was detected before death.

Case 2: In 1923, Dr. Chevalier Jackson reported the case of a 48-year-old white male who worked as a clerk in a general store. Hoarseness, which began two years previously, became progressively worse, and dyspnea developed. The patient was told he had pulmonary and laryngeal tuberculosis and asthma. He was unrelieved by a change in climate and, the dyspnea becoming worse in spite of epinephrine injections, a tracheotomy was performed. At another institution the patient's illness was diagnosed as malignancy, but the pathologist's report failed to confirm this diagnosis.

At the time Dr. Jackson saw and described the case there was a purplish red nodular area of inflammation of the skin about the tracheotomy wound. The patient complained of burning and soreness of the skin and noted slight cough and expectoration of a small amount of mucopurulent secretion. He had no fever, and had had slight weight loss.

The following is a digest of Dr. Jackson's description of the mirror appearance of the larynx: An infiltrative lesion involved the left side of the larynx, part of the right side, and the interarytenoid space. The left cord and the ventricular bands were not distinguishable because of the presence of a grayish, granular mass, which looked as though it had been brushed with silver nitrate. The left arytenoid was fixed, and the arytenoid half of the epiglottic fold was swollen. The right cord was replaced by a nodular mass which extended from the anterior to the posterior commissure, and was described as having a glistening "frog spawn" appearance.

Biopsy from the larynx showed inflamed and ulcerated mucosa, with considerable epithelial hyperplasia. Fibrosis, and numerous small foci containing giant cells, were present in the submucous tissue, giving "somewhat the appearance of a tuberculous lesion." Biopsy of the skin about the tracheotomy wound revealed blastomycosis. The patient was treated with potassium iodide.

Case 3: Dr. Gordon B. New reported two cases in 1928.* The first, seen in 1919, was a 51-year-old farmer who, at the time of examination, had been hoarse for 18 months. In 1902, following an attack of influenza, he had been hoarse for six months, and slight shortness of breath had persisted to the present illness. The patient complained of some cough, and aphonia had developed a few days prior to Dr. New's examination. There had been a weight loss of 35 pounds in the last two years. Chest X-ray, Wassermann, and sputum examinations were all negative.

The larynx was described as showing some scarring on both vocal cords. The glottis was small, and a small web was noted at the left vocal angle. There was a roughened area in the interarytenoid area. A small, grayish nodule was present on the right vocal cord near the middle, and both cords were somewhat inflamed and thickened.

Biopsy revealed inflammatory tissue only, with marked epithelial fibrosis. Tracheotomy was done because of dyspnea. Dermatitis developed around the tracheal opening, and a biopsy from the skin showed blastomycosis. Later biopsy of the larynx also showed blastomycosis.

The patient gained weight and improved rapidly in voice and general health on potassium iodide and X-ray therapy. The larynx remained

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Dr. New reviewed an additional case of primary fungus infection reported by Sartory;[21] this, however, is of the European cryptococcus type (and I do not include it).

somewhat scarred and thickened, but no evidence of activity of the blastomycotic lesion was noted at subsequent examinations. He was apparently well in 1928.

Case 4: Dr. New's second case was a 35-year-old iceman who, at the time of his examination on Nov. 16, 1927, had been hoarse for seven months. He described the onset as an acute cold; hoarseness and dysphagia gradually developed. The patient had had no night sweats, cough, or fever. Tuberculosis had been diagnosed by previous physicians, and repeated biopsies elsewhere had revealed only inflammatory tissue.

The chest X-ray, Wassermann and sputum examinations were negative. The larynx was described as showing an extensive chronic process involving both sides. The right false cord and the anterior part of the left were described as "mammillated in appearance," and some areas were covered by a thin grayish membrane. Both arytenoids were somewhat swollen and restricted in motion.

The biopsy, taken by Dr. New, was reported to show changes resembling tuberculosis; however, on further study, a diagnosis of blastomycosis was made.

The patient took massive doses of potassium iodide and showed marked improvement.

Case 5: In 1936, Dr. T. E. Fuller reported the case of a 71-year-old male who came under his observation on March 7, 1930. The patient dated the onset of throat discomfort to an attack of influenza suffered in January of that year. Examination on March 7 was essentially negative, except for the vocal cords. They were described as "dull," and "the picture was exactly what we see in people who have smoked to excess for a long time."

The patient was next seen in September. He was hoarse and had great difficulty in swallowing. Dr. Fuller states that "examination revealed a large cauliflower mass involving the right arytenoid, the interarytenoid space, the aryepiglottic fold, the lower part of the epiglottis, and the base of the tongue." No enlarged lymphatic glands were present. The Wassermann was negative, and no evidence of tuberculosis was noted. Dr. Fuller's clinical impression was carcinoma. A consultant agreed but felt that, because of the patient's age, irradiation with radium, rather than surgery, should be tried. This treatment was refused, and shortly gastrostomy, and later tracheotomy, was required.

By the Spring of 1931 there was swelling of the glands in the right side of the neck attributed to carcinoma; however, as fluctuation occurred, the area was incised, and pus was obtained. Several draining sinuses formed, around which there was extensive granulation. Sections were taken from the granulating areas and reported as follows: "Section shows hyperplasia of squamous cell epithelium with irregular papillary arrangement and rather incomplete differentiation, in that hyalinization is not prominent. Cells vary somewhat in size, shape, and staining qualities, and show occasional mitotic figures. The basement membrane, however, is in general sharp. The stroma is edematous and throughout infiltrated with leucocytes, lymphocytes and endothelial cells. . . ."

Cultures from the pus gave a growth of blastomyces dermatitidis.

The patient died on July 15, 1932, and a partial autopsy was done. A large retropharyngeal abscess was located, which probably extended into

the mediastinum. The pathologist reported that he could find only chronic inflammatory tissue. At a later date more sections were removed from the gross specimen, however, and the pathologist reported squamous cell carcinoma Grade IV.

Dr. Fuller reports this case as a "low grade of carcinoma which probably began in the postcricoid region and that the infection by the blastomycetes was secondary, producing the deep abscess and the toxemia from which he died. On the other hand, it is possible that the blastomycosis may have been primary, and that the carcinoma resulted from the chronic inflammation."

COMMENT.

Difficulty has been encountered in making a diagnosis of blastomycosis from laryngeal biopsy.

In Case 1, alone of this group, the patient gives a history of probable direct exposure to blastomycosis—*i.e.*, the rotting, decaying material of the grain elevator. Although the patient was first clinically diagnosed as having tuberculosis, in this case the laryngeal biopsy was immediately diagnosed as blastomycosis.

In Case 2, and in Case 3, in both of which the laryngeal biopsies had been inconclusive, the biopsy diagnosed as blastomycosis was obtained from skin lesions surrounding the tracheotomy wound. In Case 3, a later laryngeal biopsy, taken after that of the skin lesion, was also considered to be positive for blastomycosis.

In Case 4, as given by Dr. New, the biopsy was diagnosed as blastomycosis only after considerable study, while repeated biopsies elsewhere revealed only inflammatory tissue.

It would be extremely interesting to re-examine the slides of Case 5. While it is possible that blastomycosis and Grade IV carcinoma coexisted, the blastomycosis was undoubtedly present, and almost undoubtedly primary. Since blastomycosis may produce epithelial hyperplasia which may be confused with cancer, it seems at least possible, in the light of later cases, that blastomycosis itself was the sole lesion.

In three of these five cases the authors' descriptions of the clinical appearance of the larynx greatly resemble the description considered typical for cancer.

Case 2 is described by Dr. Jackson in such terms as "infiltrative lesion," "nodular mass" and "fixed arytenoid," all terms clinically descriptive of cancer. Indeed, malignancy had been the clinical diagnosis of another institution.

Case 3 is described as having "a small grayish nodule present on the rignt vocal cord" and the vocal cords are described as being "inflamed and thickened."

The lesion in Case 5 is described as "a cauliflower mass" and Dr. Fuller's diagnosis was that of carcinoma.

The clinical description of Case 1 and of Case 4 most closely resembles tuberculosis.

Both Dr. New and Dr. Jackson noted a grayish appearance in the blastomycotic lesions. Dr. Jackson described the lesion as "looking as though brushed with silver nitrate." This may possibly be a helpful differentiation feature in the diagnosis of blastomycosis.

REPORT OF TWO CASES IN WHICH MISTAKEN DIAGNOSIS WAS MADE.

Case 1: On Nov. 24, 1947, a letter was received referring a 58-year-old white male cabinet maker as a service patient to the ear, nose, and throat clinic of the Duke Hospital. The patient, however, failed to present himself until Dec. 2, 1947, at which time he was examined in the outpatient dispensary by a member of the resident staff. The referring physician had made two previous biopsies of the patient's larynx. He sent us the pathologist's report of the second biopsy, which was diagnosed as squamous cell carcinoma.

At the first visit, the patient gave a history of having been perfectly well until two months before, at which time he had developed bronchitis and laryngitis accompanied by a tickling sensation in the throat, and a marked desire to cough. He had been unable to speak above a whisper for the past six weeks and recently had noticed slight dysphagia. Mirror examination was noted as showing a normal epiglottis. There was some restriction of motion of the right vocal cord, and slight edema of both arytenoids and ventricular bands. The anterior one-half of each vocal cord was obscured by the swelling, and the remaining portions were noted to be ragged in appearance. A few "shotty," freely movable cervical lymphatic glands were palpated in the neck. The remainder of the nose and throat examination was negative, except for dental infection. Kahn, Kline, and Mazzini tests taken at this examination were reported negative.

The patient was given an appointment for hospital admission, but failed to return on the specified date. Finally, on Dec. 17, 1947, his admis-

sion was completed. Physical examination, including chest X-rays, proved negative, and on Dec. 18, 1947, in my absence, after consultation with another staff physician, the resident physician performed a total laryngectomy.

I saw the patient during his convalescence, which was uneventful. Primary healing of the wound was obtained, and the patient was discharged on Dec. 27, 1947, the ninth postoperative day. The patient's temperature on the evening of admission was recorded as 100.2° F. On the morning of operation it was 97.8° F., and rose to a maximum of 101° following operation. It returned to normal on the fifth postoperative day. Penicillin in a dosage of 20,000 units was given every three hours for the first six days.

At the time of operation the larynx was submitted to the pathologists at Duke Hospital. The following quotations are from their report on gross and microscopic pathology: "The specimen consists of a larynx; the mucosal covering of the larynx is involved in a leucoplakic lesion which has irregular surfaces. The vocal cords cannot be distinguished due to edema and to tumor involvement.... All sections show essentially the same picture. The epithelium shows some down growth in places, but there is a normal maturation pattern as one progresses from the basal layer to the surface. This lesion is interpreted as a hyperplasia of the epithelium. Within the epithelium in some places there may be seen small abscesses, and occasionally one may see giant cells. In many of the giant cells . . . there are refractile, double-contoured bodies" (see Fig. 4).

The previous biopsy slide was obtained from the original pathologist and was reviewed. All sections showed the same lesion, and on Feb. 15, 1948, the diagnosis was altered from cancer to blastomycosis (see Fig. 5).

The patient was promptly readmitted on Feb. 19, 1948. He had had an uncomplicated course, except for the appearance of a red, indurated skin lesion the size of a 25 cent piece, located on the left side of the face in front of, and below, the ear (see Fig. 6). Consultation was obtained with the dermatologist, the immunologist, and the Roentgenologist. Scrapings from the skin lesion revealed blastomycetes. A biopsy of the skin lesion was diagnosed as chronic dermatitis, since neither blastomycetes nor other characteristic changes could be identified. Both skin test and complement fixation test were negative.

The patient was started on standard courses of killed blastomyces vaccine 1:1,000 Each standard course consists of the following injections measured in cubic centimeters: 0.1, 0.2, 0.4, 0.6, 0.8, 1.0, given twice weekly. The skin lesion increased in size, and the standard course of vaccine was repeated. On May 6, 1948, the skin test and the complement test were again negative, and a standard course of 1:100 vaccine was started. Since that time the patient has been given numerous standard series of vaccine and, since May 26, he has been under almost constant treatment with potassium iodide. X-ray therapy was first given over the lesion near the left ear, and was continued as other areas of skin involvement developed. In spite of treatment, the skin lesion has spread (see Fig. 7); however, the complement fixation test, the skin test, and the chest X-ray remain negative.

Case 2: A 49-year-old white male druggist, referred by Dr. Alan Davidson was first seen by me on Sept. 7, 1948. The patient's only complaint was of hoarseness, which had been present since February, 1948. He

gave a family history which was negative for tuberculosis, cancer, and diabetes. He was a man of moderate habits, and smoked less than 20 cigarets daily. His only illnesses had been those usual in childhood.

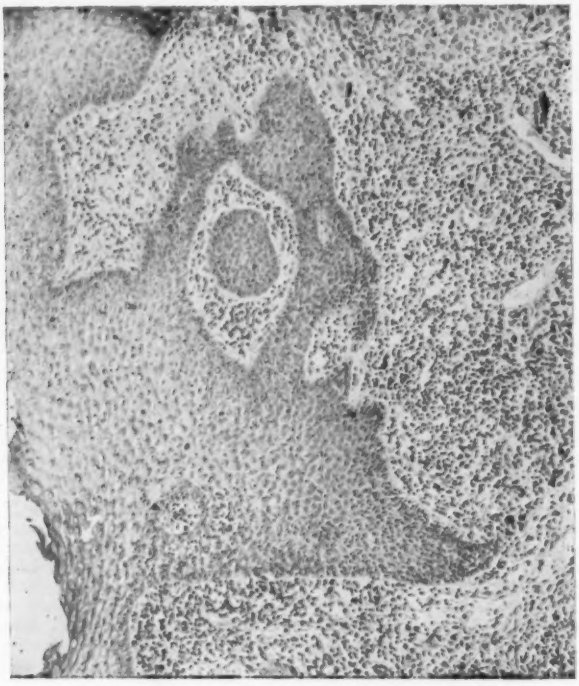

Fig. 4. Case 1. Moderate epithelial hyperplasia.

The patient dated the onset of his present illness to two severe colds; the first of these cleared completely, but the second left him permanently hoarse. The following description of the earlier appearance of the larynx was obtained from the referring physician. When first seen by him five months after the onset of the hoarseness, there was, according to his statement, "a papilloma of the anterior third of the vocal cord, as well as a mass on the anterior surface of the trachea just below the vocal cord. This mass had a whitened, ulcerated area on top of it, and it appeared clinically to be malignant; however, a biopsy taken directly from it was reported back as papilloma."*

*This biopsy, and a second specimen obtained by the referring physician in July, 1948, were both re-examined by the original pathologist, and no evidence of blastomycosis could be found; his diagnosis remained squamous papilloma.

The above quotation is taken from a letter dated Aug. 12, 1948. At the time of my examination of the patient on Sept. 7, 1948, he appeared comfortable, cooperative, and was ambulatory. He was a moderately spare, middle-aged white male, who had a hoarse voice. Other than throat lesions, no physical abnormality was noted. His temperature was 98.4°. Laboratory data was as follows: Chest X-ray, negative; serologic reactions, negative; hemoglobin, 85 per cent; R. B. C., 4,110,000; W. B. C., 8,800. Mirror examination of the larynx showed the epiglottis to be normal in appearance. The arytenoids were not swollen, but the left was

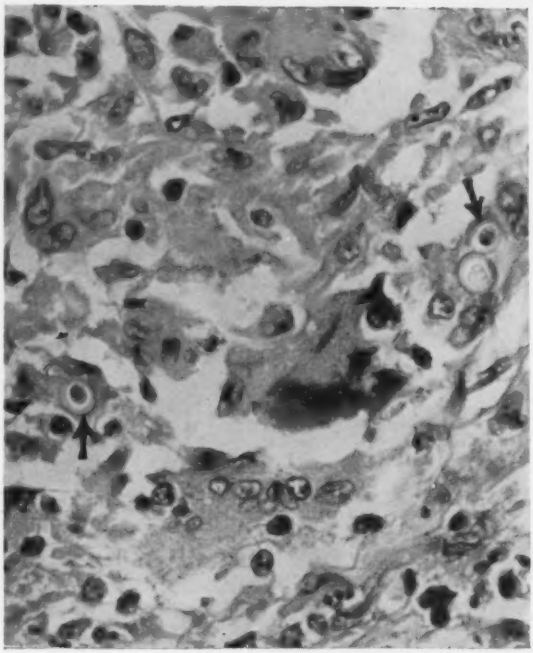

Fig. 5. Case 1. Double-contoured, refractile blastomycetes.

somewhat restricted in motion. Both vocal cords were diffusely affected by an irregular hyperplasia, and appeared reddened, thickened, and irregular. The left cord was definitely more involved than the right. No lesion was noted in the trachea at this examination. Palpation of the neck revealed one small, soft lymph node over the area of the cricothyroid membrane.

The patient was admitted to McPherson Hospital on Sept. 7, 1948, and direct laryngoscopic examination was performed on the following day.

Careful examination of the larynx at that time confirmed the mirror findings. The mucosa below the level of the vocal cords appeared smooth, and I was unable to demonstrate any tracheal lesion. A biopsy specimen was obtained from the middle of the left cord and was submitted to our regular pathologist for microscopic study.

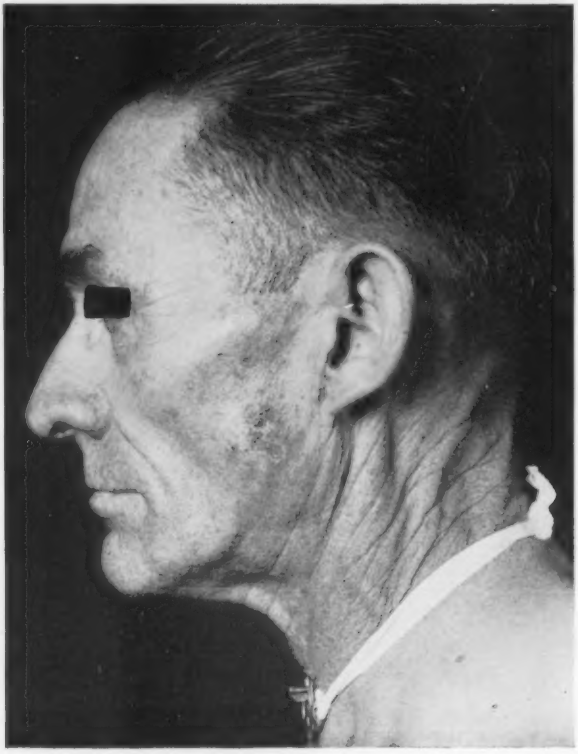

Fig. 6. Case 1. Early blastomycosis of skin.

My impression following this examination was that the lesion was not a benign papilloma. It appeared to be diffuse, invasive, and hyperplastic—probably carcinoma. Since the biopsy taken at the time of examination was diagnosed as cancer, we felt that total laryngectomy was indicated (see Fig. 8).

The patient was discharged from the hospital for a brief interval, and was readmitted on Sept. 12, 1948. Total laryngectomy was performed on Sept. 13. The soft lymph node previously felt at the cricothyroid mem-

brane level together with a wedge of tissue from the thyroid isthmus was excised. The hyoid bone was divided in the midline. The trachea was incised between the first ring and the cricoid cartilage, and the larynx was dissected from below, upwards. The larynx, including the epiglottis,

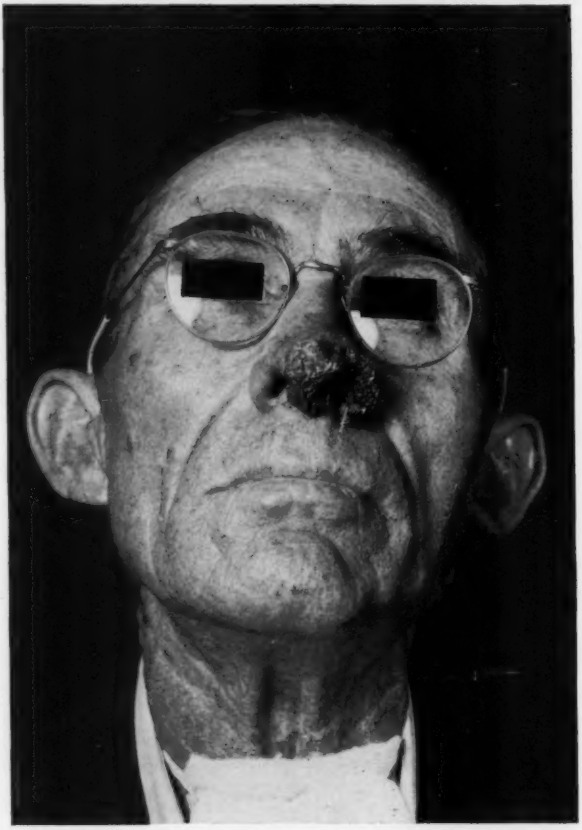

Fig. 7. Case 1. Showing spread of skin blastomycosis.

was excised, together with the pre-epiglottic tissues. These structures were inspected, and seemed to include all the growth; however, when the tracheal stump was examined, the mucosa seemed thickened, with a surface which was faintly granular and somewhat reddened. Because of these abnormalities, I took a specimen of the tracheal mucosa for examination (see Fig. 9).

The usual double row closure of the pharynx was carried out, and the constrictor and ribbon muscles were brought together to reinforce the closure.

The patient's postoperative course was entirely uneventful. Primary healing was obtained, and the patient left the hospital on the eleventh postoperative day. The highest temperature elevation of 100.6° F. occurred on the second day, and the temperature was normal from the sixth

Fig. 8. Case 2. Marked epithelial hyperplasia suggesting cancer.

day onward. The patient was given a daily injection of 300,000 units of procaine penicillin during his stay in the hospital. On the third postoperative day the pathologist reported the lesion to be blastomycosis rather than cancer, and we added 1 gm. of streptomycin to the daily treatment.

After discharge from the hospital the patient was immediately referred to Dr. D. T. Smith, Dr. Robert Reeves, and Dr. N. F. Conant for treatment of blastomycosis, and is still under active treatment. Immediately

after his discharge his immunologic reactions were determined. The skin and the complement fixation test were both negative. On Oct. 9, 1948, the patient started a standard course of blastomyces vaccine injections, which were given him by his family physician. He returned for further tests on Nov. 12, 1948. He had gained weight, and the local appearance

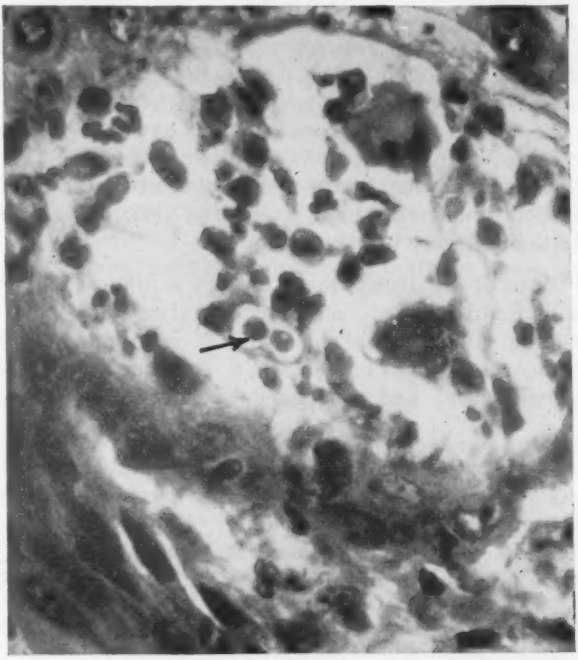

Fig. 9. Case 2. Budding blastomycete.

of the tracheotomy and of the pharynx was normal. The standard series of vaccine was repeated. By Jan. 1, 1949, the patient noted bloody cough, and examination revealed extensive granulation of the upper trachea, with narrowing of its lumen. Potassium iodide was added to the treatment, and three X-ray treatments of 100 r. each were given over the tracheal opening. Marked improvement was noted.

At the present time, slightly more than two years after operation, this man seems to be in relatively good health; he is working, and has maintained his weight. He has developed an excellent pharyngeal voice; his main complaint is of occasional coughing of bloody mucus from the trachea. Chest X-rays have been repeatedly negative. He is at present taking blastomyces vaccine and potassium iodide. Unfortunately, he has

not been able to tolerate large doses of potassium iodide, and frequently has to discontinue it. His last complement fixation test, taken Aug. 9, 1950, was positive in a dilution of 1:4, while the skin test remained negative. This may possibly signify his immunologic reclassification in the unfavorable anergic group.

SUMMARY.

Both the cutaneous and systemic forms of blastomycosis, though rare, are found widely distributed in the U. S. A. and Canada. This paper deals more fully with the less common and far more serious systemic variety, which as shown by the literature, is often mistaken for tuberculosis. Errors in diagnosis may make it seem more rare than is the actual case, for the recognition of one case in a given area sometimes seems to lead to the discovery of others.

All forms of the disease are caused by a fungus, the blastomyces dermatitidis. In tissue, this organism reproduces only by budding. When incubated at 37° C. it produces a yeast-like colony composed of budding cells. At room temperature, a filamentous colony and round and pyriform conidia may be found.

The pathologic changes noted in tissues affected by blastomycosis vary greatly. Giant cells, sometimes containing double-contoured refractile, budding blastomycetes, caseous necrosis and epithelial hypertrophy may vary in extent so that confusion in diagnosis often exists.

A skin test with blastomyces vaccine, and a specific complement fixation test aid in determining the patient's immunologic state and are a valuable guide in treatment and in prognosis. Blastomycosis is resistant to all recent antibiotic and chemotherapeutic agents and responds best to potassium iodide, X-ray, and possibly surgery in certain selected instances.

The similarities between systemic North American blastomycosis and tuberculosis have been frequently pointed out in the literature, while the close similarity of the primary laryngeal form to laryngeal cancer has not. Well-trained pathologists of various institutions have experienced difficulty in arriving at a positive diagnosis of this disease.

CONCLUSIONS.

1. Primary laryngeal blastomycosis may present laryngeal changes clinically indistinguishable from laryngeal cancer.

2. Small biopsy specimens, such as those obtained from the larynx, rarely permit a positive diagnosis of blastomycosis and the epithelial hypertrophy, when marked, may lead to an erroneous diagnosis of cancer.

3. The two patients subjected to laryngectomy are still alive three and two years, respectively, after operation; however, they still show evidence of blastomycosis and, in my opinion, surgical removal of seemingly localized infection should be undertaken only after a thorough study and an ample trial of the usual vaccine, potassium iodide and X-ray treatments has failed to produce cure or marked improvement.

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THE SURGICAL MANAGEMENT OF CARCINOMA OF THE PARANASAL SINUSES.*

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At the present time the most effective treatment for carcinoma of the paranasal sinuses consists in utilizing a combination of radiation and surgery. There is some disagreement, however, as to how these agents should be used together.

Some have advocated the initial use of cancer-lethal doses of X-ray or radium followed by a surgical procedure to provide drainage for the sloughing tumor mass. Windeyer,[1] for example, gives deep X-ray therapy followed in four weeks by surgical removal of the palate and alveolus. Some of his patients are treated further by intracavitary radium. Tod[2] advocates the implantation of a radium tube into the center of the neoplastic mass and uses surgical drainage when necessary. Prolonged cures resulting from deep X-ray therapy alone have been reported by Baclesse,[3] del Regato,[4] Lenz[5] and others. Radiation therapy is not without its complications. When given in effective dosage there is necrosis of soft tissue and often of bone. Surgical drainage is then required to prevent serious sepsis. Bronchopneumonia and meningitis which were serious complications in the past can now be controlled with antibiotics. The threat of ocular complications is great when the ethmoid cells have to be irradiated.

There are also those who prefer an initial surgical procedure followed by radiation. Ohngren[6] and New[7] have used surgical diathermy to destroy the maxilla and its contents. This is followed by deep X-ray or intracavitary radium in most cases. Hautant *et al.*[8] and Collins and Poole[9] have used

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moderately extensive surgical resection of the maxilla and followed with intracavitary radium. Schall[10] and others perform a wide radical removal of the maxilla and adjacent structures and follow with radiation. Wille[11] reported on 220 patients treated at the Norwegian Radium Hospital and concluded that the best therapeutic regime for carcinoma of the maxilla was surgical removal followed immediately by radiation therapy.

It is known that cancer-lethal radiation therapy is followed frequently by sepsis and sequestration and that wide surgical exposure and drainage are necessary. Thus, because eventually some form of surgery is required following radiation therapy, it seems logical that we should resort to surgery in the beginning and that this surgical procedure should be a vigorous attempt to remove the neoplasm completely. If this is followed by cancercidal doses of radiation we have done the most for our patient.

It is realized that a rigid therapeutic regime cannot be applied to all patients. In the first place, 5 to 10 per cent of all paranasal sinus neoplasms are sufficiently radiosensitive to justify the primary use of radiation. Lymphosarcoma and transitional cell carcinoma fall into this group. Margaret Tod,[2] of the Holt Radium Institute, reports 50 per cent five-year survivals of 18 cases of lymphosarcoma treated by X-ray therapy alone. Primary surgical resection should not be attempted when there is no reasonable chance for complete removal of the neoplasm. The criteria for operability of a particular lesion are determined to some extent by the surgeon's skill and courage. There are several contraindications to the definitive surgical removal of a primary malignancy of the paranasal sinuses. *1.* Complete resection is impossible when there is invasion of the base of the skull. *2.* If the neoplasm is adherent to the walls of the nasopharynx it probably cannot be resected. *3.* Surgery is to be avoided if there is extensive bilateral involvement because of the hopeless facial defect which results. *4.* Distant visceral or skeletal metastases almost certainly preclude a cure and complete resection of

the primary should not be attempted. *5.* The same holds true when there are insurmountable regional lymphatic metastases. In our experience about 30 per cent of all patients with carcinoma of the paranasal sinuses fall into an inoperable group. Such patients should receive palliative radiation therapy. Palliation for two or three years is not unusual and five-year survivals have been reported. After excluding those patients with advanced lesions and those with radiosensitive tumors, there remains a group (comprising 60 to 70 per cent of the total) having squamous cell carcinoma or adenocarcinoma in which excision is possible.

A biopsy must be taken to establish the histological type before treatment is begun. It must be deep enough to avoid necrotic surface tissue. Bleeding may be active but is readily stopped with gelfoam and pressure packing. A needle biopsy taken through the face is to be avoided because of the possibility of creating a suppurating sinus tract and the danger of implanting tumor cells into the facial tissues.

Benign tumors need not be treated in the manner to be described here for malignancy. The mixed tumor and adamantinoma, for example, are inherently benign. It is well known that they recur after incomplete excision and then may be locally destructive and invasive. Although these tumors must be completely removed, the palatodental plate, zygoma, orbital contents and nasal bones often can be preserved. A simple lateral rhinotomy incision (without splitting the lip) or a wide Caldwell-Luc incision usually is adequate for removal of these tumors. The surgical procedure should always provide for a permanent large infralabial fenestra. A soon as the packing has been removed (three to five days) the patient should be provided with a prosthetic appliance for the fenestra as seen in Fig. 1. Such a fenestra permits thorough postoperative examination and makes possible the early detection and treatment of recurrences.

All patients with suspected malignancy of the paranasal sinuses should have Roentgen studies of the skull, including an axial view. In the search for metastases there should be a

routine preoperative chest Roentgenogram. Patients who have had nasal hemorrhages may require a preoperative blood transfusion. Depleted plasma proteins can be corrected by administering whole blood or plasma transfusions. Because most carcinomas of the nose and paranasal sinuses are infected, it is important to administer antibiotics preoperatively for four or five days in dosages sufficient to insure an adequate blood level.

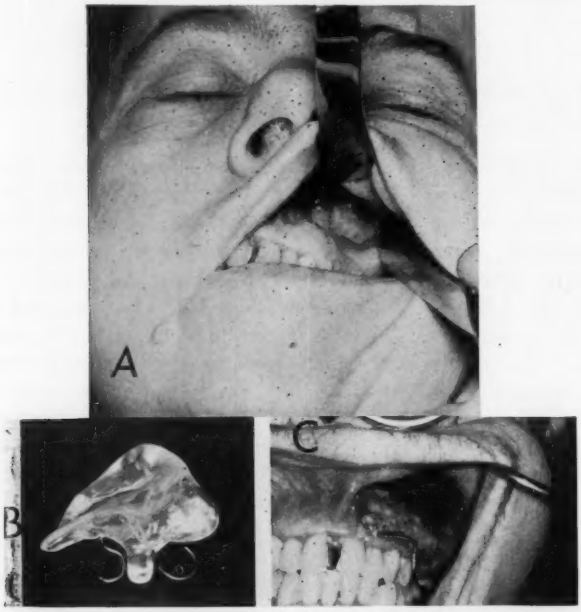

Fig. 1. (Mrs. H.) Removal of maxilla but with preservation of the palatodental plate (for adamantinoma). (a) View showing the infralabial fenestra. (b) The prosthetic appliance. (c) The prosthetic appliance in place.

Whether the surgeon uses diathermy or cold steel to perform the resection is not a critical factor. It is important by either method to achieve a wide surgical exposure and to perform a complete resection of the area involved.

If the external carotid artery on the same side as the lesion is ligated first, there is less bleeding during the operation, thus giving a clearer operative field. One to three units of whole blood are usually required during surgery to replace blood loss.

Intravenous pentothal plus intratracheal nitrous oxide-oxygen provides satisfactory anesthesia. A long gauze strip is placed in the pharynx around the intratracheal tube to prevent aspiration of blood and pharyngeal secretions.

BLOCK RESECTION OF THE MAXILLA.

Block resection of the maxilla may be performed in the following manner:

The incision begins midway between the inner canthus and the bridge of the nose as seen in Fig. 2a. It follows the lateral border of the nose, circumvents the nares and extends through the entire thickness of the upper lip in the median or paramedian line. The superior labial and lateral nasal branches of the facial artery must be tied or coagulated during this procedure. An incision is made through the mucosa deep in the gingivolabial fold from the midline to the maxillary tuberosity. A facial flap is elevated so as to leave periosteum, fat and connective tissue on the anterior surface of the maxilla. The lateral wall of the nose is separated from its attachment to the margins of the pyriform aperture, thus exposing the interior of the nose (see Fig. 2, b and c).

The alveolus and bony palate must be removed with the resected block. The mucoperiosteum of the roof of the mouth may be preserved if it has not been invaded by cancer. This membranous flap can be separated from the bone with a heavy mastoid elevator, beginning on the buccal surface of the alveolus (see Fig. 3a). It is necessary to proceed with caution because the periosteum is firmly adherent and perforation can occur easily near the midline where the mucoperiosteal layer is thin. When teeth are present the palatal flap may be elevated from an incision made on the lingual surface of the alveolus

(see Fig. 3c). If the neoplasm has involved the palate, the entire thickness must be removed well beyond visible tumor so as to insure complete excision (see Fig. 3b).

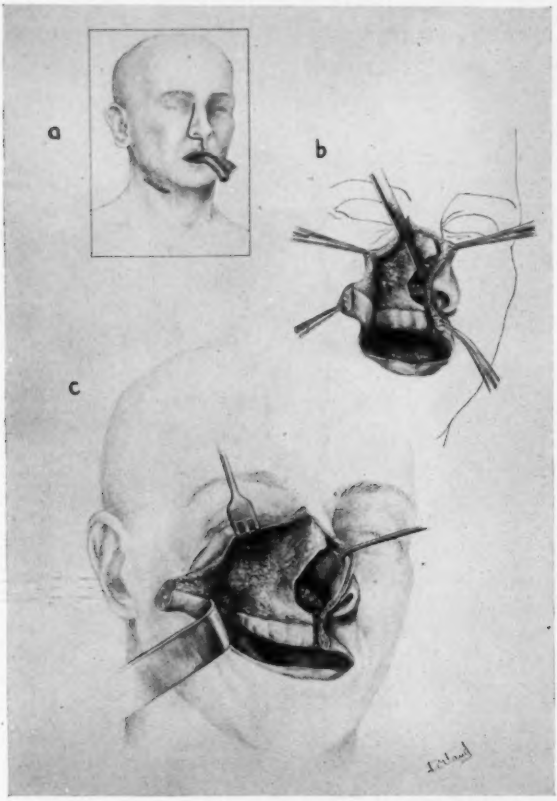

Fig. 2. Block resection of the maxilla. (a) Incision. (b and c) Exposing the anterior surface of the maxilla and the interior of the nose.

The nasal bone and the frontal process of the maxilla should be removed in most cases. This removal gives wide access to the ethmoidal labyrinth, nose and frontal sinus. If it has been established beyond doubt that the malignancy has involved

only the lower part of the maxilla, the frontal process of the maxilla may be divided in its midportion, thus preserving the nasel bones (see Fig. 3, d, e, f).

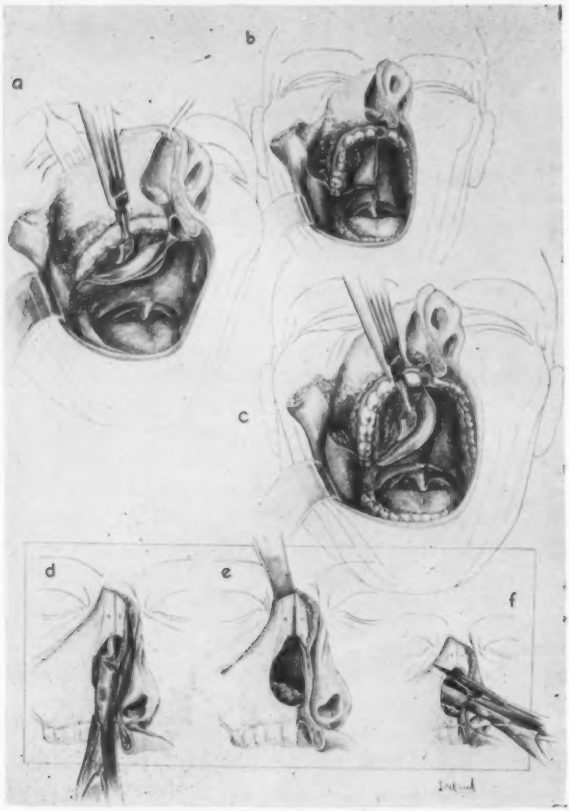

Fig. 3. Methods for handling the palate and nasal bone. (a) Elevation of a mucoperiosteal flap from the alveolus and hard palate when the palate has not been invaded by cancer. (b) Hemisection of the palate when these tissues are involved by neoplasm. (c) Elevation of the flap when teeth are present. (d) Separation of the nasal bones in the midline. (e) Cutting through the nasal bone and the frontal process of the maxilla. (f) The alternative method of cutting through the midportion of the frontal process of the maxilla is used only when the neoplasm is restricted to the inferior portion of the maxilla and therefore is rarely applicable.

A sharp osteotome or a saw is used to cut through the zygomatic process of the maxilla (see Fig. 4a). The periosteum is next elevated from the floor of the orbit in its anterior one-half.

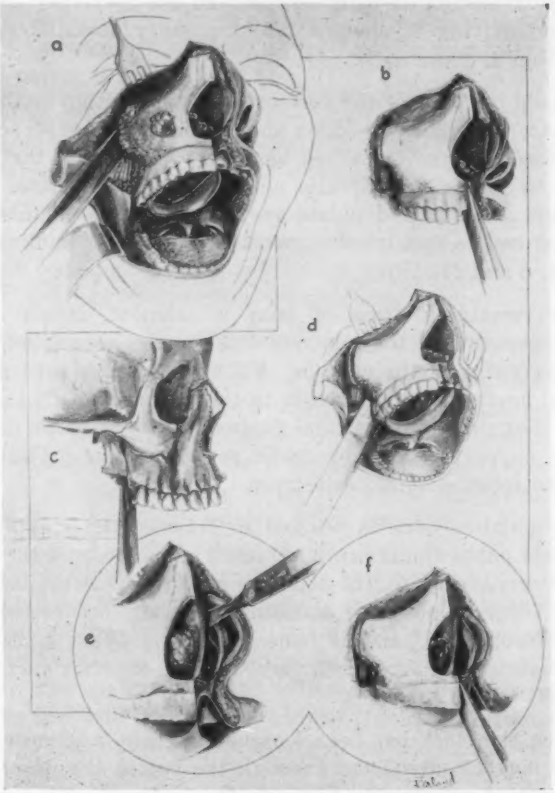

Fig. 4. Cutting through the zygomatic process of the maxilla (b) Dividing the alveolus and palate near the midline. (c) View of skull to show the relationship of the pterygoid processes to the posterior wall of the maxilla. (d) The osteome is placed behind the tuberosity of the maxilla and driven up into the pterygomaxillary fossa. (e) When the nasal septum and floor of the nose are invaded by neoplasm, the septum is divided anteriorly, thus exposing the opposite nasal meatus. (f) The alveolus and palate are divided through the floor of the nose on the opposite side.

The alveolus and bony palate are divided near the nasal crest with an osteome as shown in Fig. 4b.

The posterior surface of the resected block must be separated from the sphenoid bone. The palatine bone, which becomes part of the specimen, is separated from the pterygoid plates by placing an osteotome behind the maxillary tuberosity and driving it upward into the pterygomaxillary fossa (see Fig. 4, c and d).

Should the floor of the nose or the nasal septum be invaded by cancer the block resection should include those structures. The nasal septum is incised vertically anterior to the lesion so as to expose the opposite nasal meatus. The nasal bones and the alveolus and palate are divided in the manner described before, but on the opposite side of the midline (see Fig. 4, e and f).

The remaining areas of bony attachment usually break spontaneously. If they do not, this can be accomplished by gentle rocking of the maxilla. A fracture occurs through the orbital surface of the maxilla to the paper plate of the ethmoid. Sagging of the orbital contents will not occur because of the support of the zygomatic portion of the orbital floor and the intact orbital periosteum.

The palatine bone is wedged in between the maxilla and sphenoid bones and is firmly attached to the body of the sphenoid bone; therefore, the separation of the resected block of tissue from its posterior attachment usually occurs by fracture through the palatine bone. Thus the upper portion of the palatine bone remains attached to the anterior surface of the body of the sphenoid.

When the block has been loosened slightly, a narrow osteotome may be introduced through the cut in the nasal bone and carried posteriorly through the ethmoid cells, on the under surface of the orbital plate of the frontal bone. On reaching the anterior surface of the body of the sphenoid, the entire mass is displaced downward. When the nasal septum has been included in the resection, the perpendicular plate of

the ethmoid is also divided along its superior attachment. A finger may be introduced into the nose to aid in delivering the resected block of tissue, as shown in Fig. 5a.

To control bleeding, hot towels are applied with pressure. The internal maxillary artery is grasped and tied. The smaller vessels may be coagulated. Remaining fragments of the ethmoidal labyrinth are curetted away. The sphenoidal sinus may be evacuated after removing its anterior wall. The final surgical defect is seen in Fig. 5b.

To aid in preventing the implantation of cancer cells, the area should be thoroughly irrigated with saline and the coagulating current used on all denuded areas.

Follow-up examinations for cancer of the maxilla and nose cannot be made satisfactory through the nostril or through a small oral opening. A large infralabial window must be constructed and kept open with a prosthetic appliance.

The window may be constructed in the following manner:

The labial mucosa is undermined and transfixed upward (see Fig 5, c and d). The anterior margin of the palatal flap is inspected for viability. The portion of this flap which was elevated from the alveolar process is thick and avascular and may have to be sacrificed. The anterior margin of the palate can be folded upon itself and sutured down (see Fig. 5, d and e). A palatal flap so constructed provides a pliable rim of soft tissue which allows easy introduction and removal of a prosthetic appliance without sacrificing its stability. Only the posterior end of the oral incision is sutured, so as to leave a window at least 4 cm. long. Gelfoam strips are laid on all raw surfaces. The cavity is packed with vaseline gauze strips in a manner that will allow easy removal from the oral window or nostrils. The subcutaneous layers of muscle and fascia are brought together with interrupted absorbable sutures. The skin is closed with fine silk sutures. The vermilion border of the lip should be accurately approximated (see Fig. 5f).

A head dressing is applied with moderate pressure over the operated area. Antibiotic and chemotherapeutic agents are

used in doses sufficient to insure adequate blood levels for five to 10 days. The outside dressing is changed daily. The packing is left undisturbed for five days unless the patient develops an elevated temperature; in that case it must be removed earlier. The sutures may be removed on the fifth or sixth postoperative day. The cavity should be irrigated twice daily with warm, normal saline solution after the fifth day.

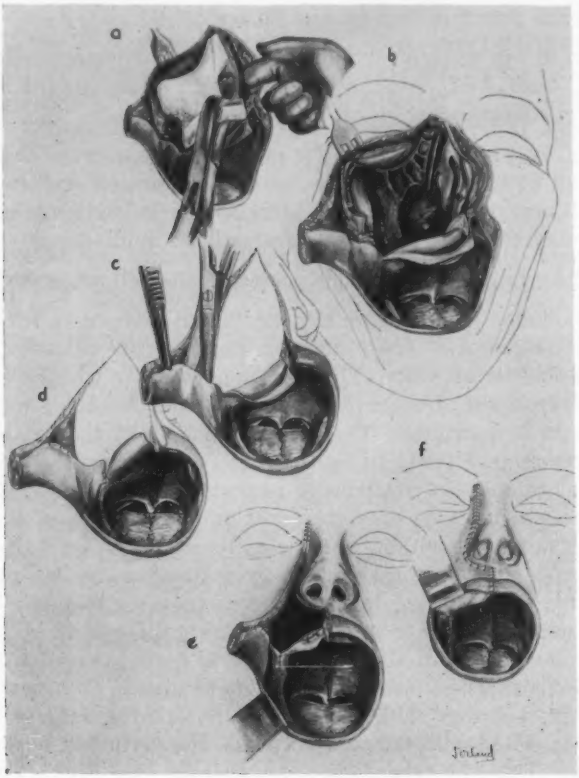

Fig. 5. (a) Delivering the block of tissue. (b) View of surgical defect. (c and d) The mucosa on the inner surface of the lip is undermined and transfixed upward. (e) The anterior margin of the palate is folded over on itself and sutured down. (f) Closure completed, leaving a large infralabial fenestra.

A prosthesis is constructed as soon as the gauze packing is removed. The appliance serves the following functions: *1.* It fills the infralabial opening and prevents contamination of the operative cavity with food and secretions. *2.* It prevents cicatricial stenoses of the window. *3.* It prevents deformity of the face by supporting the lip, nasolabial angle and cheek, and *4.* it provides a masticating surface (see Fig. 6).

When it has been possible to save a mucoperiosteal flap from the under surface of the hard palate, an elastic rim of tissue is provided along the medial and posterior margins of the window. Thus, the bulb of the prosthesis is constructed with a little overhang so that when it is introduced it will snap into place and thus be held firmly.[13]

The patient may leave the hospital between the seventh and tenth postoperative day. After two to three weeks all patients should receive intracavity radium or deep X-ray therapy. The patient continues to irrigate the cavity twice daily with normal saline solution until crusting and discharge have ceased, after which the irrigations are necessary once daily for an indefinite period. The patient should return for careful cleansing of the area at biweekly or weekly intervals over a period of two or three months until epithelialization of the cavity has occurred.

BLOCK RESECTION OF THE MAXILLA, ZYGOMA, AND CONTENTS OF ORBIT.

Proptosis almost always means that invasion of the orbit has occurred and, therefore, proptosis is an indication for evisceration of the orbit. The operation leaves a serious cosmetic defect. It should be recommended only after careful consideration of the patient's psychological attitude and after evaluation of the chances for a cure. Often the patient cannot carry on a gainful occupation after this operation. Most of these patients are elderly individuals, however, who are prepared to retire from extensive occupational and social contact with the public.

In this operation the face splitting incision is used, as already described, plus an extension carried laterally around the palpebral fissure about 5 mm. from the lid margins and for a distance over the zygoma, as shown in Fig. 7a. The

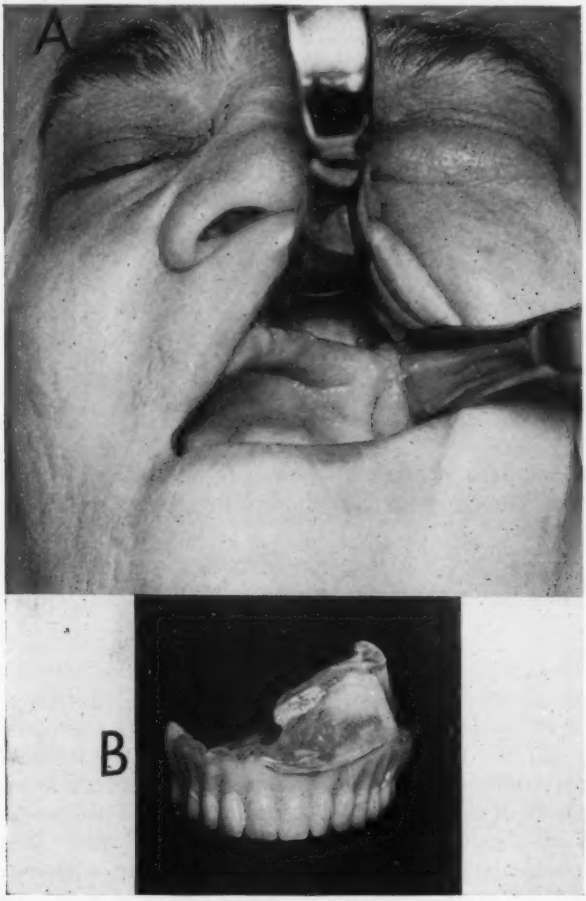

Fig. 6. (a) (Mrs. T.) View of the window six months after operation. (b) The prosthetic appliance.

facial flap is reflected, as described previously. The skin of the upper lid is retracted to the supraorbital ridge. The zygomatic bone and the orbital contents become part of the resected block in this operation. The osteotome is used to break through the temporal and frontal processes of the zygoma. The maxilla, ethmoid, labyrinth, palate and nasal septum are handled in the manner previously described. The periosteum is reflected down from the roof of the orbit and the orbital contents are displaced downward until the pedicle of the eye is exposed. The pedicle is the last attachment to be cut. It is severed with a curved scissors, and the bleeding is controlled temporarily with hot packs and pressure until the ophthalmic artery can be grasped and tied (see Fig. 7b).

The anterior and inferior walls of the frontal sinus and the supraorbital ridge may be removed as shown in Fig. 7c. Any sharply protruding bone is cut away with a bone rongeur and the bone edges are rounded. The denuded surfaces are cauterized thoroughly. The upper lid is laid as a flap onto the roof of the orbit.

The mucosal border on the inner surface of the lip is sutured to the free edge of the palate so as to completely close the mouth from the operative cavity. The facial flap is laid onto the floor of the operative defect and its free margin sutured to the upper surface of the soft palate. The lip incision is closed to the lateral margin of the nostril. The part of the incision which extended over the zygoma is closed. The completed operation is seen in Fig. 7d. No attempt is made to close the surgical defect because *1.* primary closure usually breaks down and *2.* the presence of a large permanent opening facilitates both after-care and administration of radiation therapy. The packing, dressings and after-care is similar to that already described for resection of the maxilla.

Intracavitary radium therapy or deep X-ray therapy should be given after two to six weeks. Radium therapy can be given, incorporating radium needles into a wax impression of the operative defect.

After two to three months a prosthesis may be made to cover the surgical defect. The appliance should incorporate a glass eye and restore the original appearance of the patient as far as possible. It can be made of latex rubber or some other lightweight material. Spectacles help to hold the prosthesis in place. The patient will not wear the appliance if it

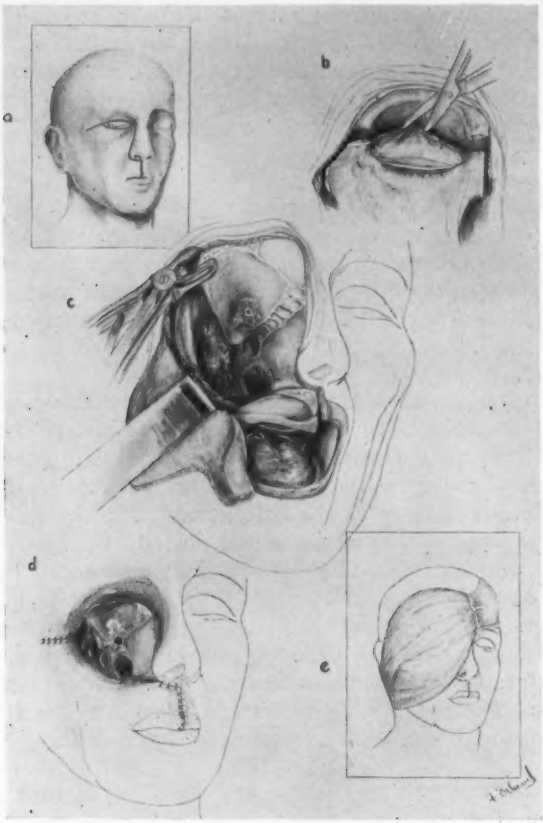

Fig. 7. Resection of the maxilla, orbital contents and zygoma. (a) The incision. (b) The orbital contents are displaced downward and thus become part of the resected block of tissue. (c) The surgical defect. (d) View at completion of operation. (e) Pressure dressing.

is heavy, ill fitting or uncomfortable. Some patients prefer to wear a surgical dressing to cover the defect even though the prosthedontist has used the utmost care in constructing a good appliance.

Fig. 8. (Mrs. B.) Left-sided proptosis indicates encroachment on orbital contents. (b) View of surgical defect on the fifth postoperative day. (c) The resected block of tissue. (d) Microscopic section through part of the specimen to show destruction of the orbital floor and displacement of the globe. (e) (Mr. R.) Surgical defect four months postoperatively. (f) With facial prosthesis.

SUMMARY.

In general, the most effective treatment for carcinoma of the paranasal sinuses consists of surgical resection followed by radiation. Exceptions are those patients with radiosensitive tumors and those having malignancies beyond any reasonable chance for complete removal.

When block resection of the maxilla, ethmoid labyrinth and nasal septum is performed, a large permanent infralabial window should be constructed. A properly made prosthetic appliance will keep this window open and will prevent deformity by supporting the tissues of the face.

If proptosis has existed, the orbital contents must be included in the resection. This operation leaves an extensive facial defect. Many patients will accept the deformity when they understand that it is necessary for a cure.

Radiation therapy should routinely follow all surgical resections for carcinoma of the paranasal sinuses.

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THE ARTICULATION FUNCTION OF PATIENTS WITH CONDUCTIVE DEAFNESS.*

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INTRODUCTION.

Otologists have long recognized that certain hard of hearing patients understand speech with relative ease when it is made loud enough to override their hearing losses. Other patients complain that they "hear" speech but do not understand it, and are helped more by slow delivery and careful enunciation than by mere increase in intensity. From these observations, the concept of two related yet distinctly different dimensions of hearing for speech has emerged. The first dimension, sensitivity, is the ability to detect soft speech. The second, discrimination, is the ability to understand audible speech by differentiating between phonetic elements. A test of sensitivity, therefore, tells us how intense speech must be made before the patient responds to it, while a test of discrimination tells us how well he recognizes words in speech which is already loud enough. In general, a simple reduction of sensitivity, a so-called "hearing-loss", has been associated with conductive deafness, while difficulty in discrimination has been related to nerve deafness.

Word lists for testing hearing for speech were compiled during the war at the Psycho-Acoustic Laboratory of Harvard

*From the Hofheimer Audiology Laboratory, Department of Otolaryngology, Washington University, and Central Institute for the Deaf. This work was begun under contract Noonr-272 between the Office of Naval Research and Central Institute for the Deaf. It was continued under contract V1001M-577 between Veterans Administration and Central Institute for the Deaf, later transferred to the Hofheimer Audiology Laboratory at McMillan Hospital (Washington University). The work there is supported by a grant from the Hofheimer Foundation.

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University.[1] Spondaic (disyllabic) words such as "railroad" and "stairway" were chosen, since they were found to be of high and approximately equal intelligibility. A test of discrimination, on the other hand, should present the difficulties encountered in understanding everyday English. The speech which we hear varies in intelligibility partly because some phonetic elements are harder to identify than others. The phonetic composition of English was therefore taken as the basis for construction of this test material. Twenty "phonetically balanced" lists of 50 monosyllables each were compiled at the Psycho-Acoustic Laboratory,[1] each list containing the phonetic elements in approximately the proportion in which they occur in English.

The general procedure in testing sensitivity with spondaic words is to attenuate the stimulus until a level is determined at which the patient is able to repeat 50 per cent of the words correctly. This level is called his "threshold for speech" and when compared with a threshold for normals obtained under the same conditions, provides a measure of loss in sensitivity in decibels. In testing discrimination, a list of monsyllables is presented at a constant intensity, and the patient's "score" is the percentage of words repeated correctly at that level.

If a series of phonetically balanced ("PB") word lists is given to a patient at intensity levels successively farther above his threshold for speech, his scores may be plotted against physical intensity (in decibels). As the intensity is increased he understands more and more of the words, until, with sufficient intensity, the test material becomes maximally intelligible for him. Such a curve is called an "articulation function".[1] In 1946, Walsh and Silverman[2] advanced the theory that the shape of the articulation function for the hard of hearing person depends upon the type of his hearing loss. They published sample articulation functions obtained on three patients whose hearing losses were, respectively, of the conductive, mixed and nerve types. At intensity levels well above threshold, the patient with conductive deafness could repeat nearly all the PB words correctly, but the patients with nerve degen-

eration could not. Walsh and Silverman suggested that such articulation tests may, therefore, be useful in diagnosis and prognosis for the fenestration operation.[cf. also 3]

The intelligibility of speech depends not only on its loudness and phonetic content, but also on the voice and enunciation of the speaker. No two speakers say a word in exactly the same way, and one speaker does not produce identical acoustic patterns as he repeats the same word. In many hearing clinics hearing for speech is tested by live voice. At Central Institute for the Deaf, however, in order to obtain standardized results, recordings of eight of the phonetically balanced word lists were made by Harrison in the Technisonic Laboratory at Central Institute for the Deaf, in 1945.[4] The talker was Rush Hughes, a professional radio announcer. These recordings, although made originally as a temporary expedient to test the advantages of recorded lists, have been used at Central Institute for the Deaf since October, 1945 and subsequently in the Hofheimer Audiology Laboratory at McMillan Hospital (Washington University) to test the auditory discrimination of all patients of Dr. T. E. Walsh who were candidates for the fenestration operation.

A "normal" articulation function for the PB lists is given by Walsh and Silverman (Figs. 5 and 7). These curves were only tentative, and the strong concavity upward in the lower part of the curve was not found in a more extensive study two years later (Davis, "Articulation Area", Fig. 8). Even this function rests on a rather small foundation, namely a group of ten normal subjects. The experiments were not systematically counterbalanced to eliminate learning factors and it was assumed (a) that all eight recorded lists were equally difficult and (b) that the normal function would reach 100 per cent correct at the level of average speech. In drawing the "normal" curve in question[3] we took into account the extensive data obtained at the Psycho-Acoustic Laboratory with the same lists of words.[1] The work at Psycho-Acoustic Laboratory, however, was done with live voice, not with our recorded tests. We have not studied the normal articulation curve for

the Rush Hughes recording in further detail because we have always regarded these particular recordings as temporary stop-gap items, but only recently have we started serious work on a new set of recordings to replace them. In the meantime other laboratories have obtained and employed copies of the Rush Hughes recordings. For them the present data will have a practical as well as a theoretical interest.

A somewhat similar study has recently been published by Beasley and Rosenwasser.[5] They determined separately the articulation functions for two sets of (recorded) monosyllables. One set is composed of "high-frequency words", the other of "low-frequency words". The two articulation functions were determined for 50 normal ears. The two functions were almost identical in shape (as well as in threshold value), and the authors state that "a composite average between the two curves does not differ significantly from the normal articulation curves for PB word lists as reported by Davis[6] and Thurlow.[7] The coincidence is gratifying, even though the word lists were not identical with our own. Incidentally, the lower levels of the plateaus (or maxima) of the articulation functions found by Beasley and Rosenwasser for patients with hearing losses (particularly high tone hearing losses) agree very closely with our own experience.

METHOD.

The group of patients and the routine of testing have been described in full elsewhere.[7,8,9] Articulation tests were conducted systematically both before and after the fenestration operation and on the unoperated as well as the operated ears. Nearly all of our articulation scores for the Rush Hughes recordings obtained on patients fenestrated without stopple enter into the present study. The series of operations extends from January, 1945 to July, 1949. They are consecutive cases except for "revisions" and also excepting four cases for whom Spanish was the mother tongue and two who failed to cooperate because of psychological difficulties.

The clinical diagnosis of nearly all of these patients was "otosclerosis". The degree of nerve involvement varied from

none (pure conductive hearing loss) to moderate (mixed deafness, predominantly conductive or "conductive-mixed") to considerable (mixed deafness, predominantly perceptive or "mixed-nerve"). No cases of pure nerve deafness have been fenestrated, and this important group is, therefore, excluded from the present study. The classification was made before the operation by Walsh on the basis of all available evidence, including history, tuning fork tests, pure tone audiometry and (in the last three years) tests of discrimination for speech. In only a very few cases was it necessary to revise the classification on the basis of additional information obtained at the operating table.

In each testing session the threshold for speech was measured by Auditory Test No. 9,[10,7] a recording of spondaic words originally prepared at the Psycho-Acoustic Laboratory of Harvard University and transcribed in the Technisonic Recording Laboratory.[4] The threshold for speech determined by Test No. 9 is the common reference point for all cases. The intensities at which the Rush Hughes PB lists were given are all reckoned as decibels above the threshold (for that ear) for Test No. 9. The hearing losses for speech range from about 20 to about 80 db.

Our routine for giving the PB tests has been changed several times. Early in the program, PB lists were given at intensity levels 10 db apart, from a level near the patient's threshold (for Auditory Test No. 9) to a high level such as 110 or 120 db re .0002 dyne/cm^2. In this way, a complete articulation function was obtained for each ear tested. Subsequently the routine was changed to include only three lists, two given at least 35 db above the threshold for Auditory Test No. 9 and one given at the patient's threshold of intelligibility for connected discourse.[11] For the latter, we later substituted a test at 85 db re .0002 dyne/cm^2 (the level of loud speech). The relationship between this intensity and the patients' thresholds for Auditory Test No. 9 varied, of course, with the hearing loss of individual patients. This procedure was followed for about one year. During the past two years,

the PB test has been given only at levels at least 35 db above the threshold for Test No. 9. By changing our routine as described above, we have given PB tests at every intensity level relative to the threshold for Auditory Test No. 9, from a few decibels below to about 60 decibels above.

Now, after five years of practical experience with these tests, we have examined the scores systematically in order to answer the following questions:

1. Are the articulation scores of the hard of hearing (using the Rush Hughes recording) correlated with clinical diagnosis, as suggested by Walsh and Silverman?

2. Are the eight Rush Hughes lists of equal difficulty?

3. What is the distribution of difficulty among individual test items in the Rush Hughes recording?

4. How reliable is the maximum articulation score ("PB-Max")?

The study indicates certain specific shortcomings of the Rush Hughes recording which should be avoided in any recordings of PB lists to be prepared in the future.

RESULTS.

The composite articulation functions (see Figure 1) show the mean PB scores, averaged at intensity levels 5 db apart, from the threshold for Auditory Test No. 9 to 55 db above it. The number of scores averaged to obtain each point on the curves was considerable (see Table 1). Actually, each point represents a range in input level of 2.5 db above and below each indicated level; that is, scores on tests given from 2.5 db below to 2.5 db above the threshold for Test No. 9 were averaged to obtain the mean score at the threshold for Test No. 9; tests given from 2.5 to 7.5 db above threshold were used for the point 5 db above the threshold for Test No. 9, and so on.

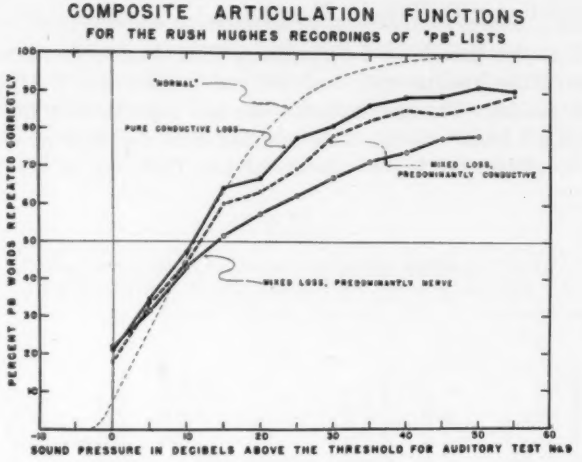
COMPOSITE ARTICULATION FUNCTIONS
FOR THE RUSH HUGHES RECORDINGS OF "PB" LISTS
PERCENT PB WORDS REPEATED CORRECTLY
"NORMAL"
PURE CONDUCTIVE LOSS
MIXED LOSS, PREDOMINANTLY CONDUCTIVE
MIXED LOSS, PREDOMINANTLY NERVE
SOUND PRESSURE IN DECIBELS ABOVE THE THRESHOLD FOR AUDITORY TEST No.9

Fig. 1.

TABLE I.

ARTICULATION SCORES OF PATIENTS WITH PURELY CONDUCTIVE HEARING LOSS.

Input Level Db Above No. 9 Threshold	No. of Tests	No. of Patients	Mean Score (Per Cent Correct)	σ*	σm*	Mean Score $\pm\sigma$	Range of Scores (Per Cent Correct)
0	97	48	21.2	12.3	1.26	8.9-33.5	0- 48
5	74	46	34.5	13.0	1.51	21.5-47.5	4- 72
10	93	41	47.1	14.1	1.47	33.0-61.2	4- 84
15	81	47	64.0	14.0	1.56	50.0-78.0	30- 90
20	84	38	66.8	16.8	1.83	50.0-83.6	42- 92
25	78	45	77.6	9.9	1.12	67.7-87.5	38- 96
30	118	54	81.0	8.4	.77	72.6-89.4	56- 98
35	229	93	86.6	7.1	.47	79.5-93.7	64-100
40	364	115	88.7	6.3	.33	82.4-95.0	64-100
45	254	87	89.1	6.2	.39	82.9-95.3	70-100
50	263	79	91.3	5.8	.36	85.5-97.1	70-100
55	155	57	90.6	6.4	.52	84.2-97.0	72-100

*σ = standard deviation of the mean.
σm = standard error of the mean.

Correlation with Clinical Diagnosis

Near the threshold for Auditory Test No. 9, the performance of the groups with and without some nerve deafness is very similar. The mean scores are *not* significantly different (by the t-test)* at the threshold for Test No. 9 or at 5 or at 10 db above it. At the threshold for Test No. 9, where all

TABLE II.

ARTICULATION SCORES OF PATIENTS WITH MIXED HEARING LOSS, PREDOMINANTLY CONDUCTIVE.

Input Level Db Above No. 9 Threshold	No. of Tests	No. of Patients	Mean Score (Per Cent Correct)	σ*	σm*	Mean Score ± σ	Range of Scores (Per Cent Correct)
0	26	16	18.0	13.7	2.74	4.3-31.7	0- 50
5	45	22	33.5	14.3	2.13	19.2-47.8	4- 70
10	39	22	44.3	16.3	2.63	28.0-60.6	16- 76
15	32	20	60.0	10.5	1.84	49.5-70.5	32- 80
20	38	19	63.1	12.3	1.98	50.8-75.4	32- 88
25	35	17	69.4	11.0	1.86	58.4-80.4	42- 86
30	65	30	78.3	7.7	.95	70.6-86.0	58- 94
35	111	37	82.6	8.5	.81	74.1-91.1	60-100
40	149	45	85.3	6.8	.56	78.5-92.1	64- 98
45	101	38	84.5	6.9	.69	77.6-91.4	62- 98
50	124	36	86.8	6.8	.61	80.0-93.6	68- 98
55	47	21	89.2	5.9	.86	83.3-95.1	76-100

*σ = standard deviation of the mean.
σm = standard error of the mean.

groups make about 20 per cent, the mean for the mixed nerve cases is actually highest, and the point at which 50 per cent of the PB words are heard correctly (the "PB threshold") is passed by all three groups between 10 and 15 db above the threshold for Test No. 9. It seems, therefore, that PB tests given at levels near the threshold for spondaic words do not

*The t-test measures the probability that the difference between two means is the result of random factors in sampling.

indicate the presence or absence of nerve involvement. Evidently the easiest PB items are just as intelligible as the spondaic words of Test No. 9, whether or not the listener has some nerve deafness. The critical items for discrimination are the 25 hardest words, and the test is most useful for this purpose when given at levels well above the threshold.

TABLE III.

ARTICULATION SCORES OF PATIENTS WITH MIXED HEARING LOSS, PREDOMINANTLY NERVE.

Input Level Db Above No. 9 Threshold	No. of Tests	No. of Patients	Mean Score (Per Cent Correct)	σ*	σm*	Mean Score $\pm\sigma$	Range of Scores (Per Cent Correct)
0	43	24	21.8	11.4	1.73	10.4-33.2	0- 46
5	53	27	31.5	11.6	1.59	19.9-43.1	8- 60
10	64	27	43.1	12.7	1.59	30.4-55.8	12- 74
15	60	27	51.7	10.8	1.40	40.9-62.5	22- 76
20	78	28	57.2	12.1	1.38	45.1-69.3	22- 76
25	65	26	62.1	10.2	1.26	51.9-72.3	40- 82
30	79	27	67.1	10.1	1.13	57.0-77.2	38- 90
35	78	23	71.4	9.4	1.07	62.0-80.8	40- 90
40	75	22	74.0	9.9	1.14	64.1-83.9	54- 88
45	39	15	77.7	13.8	2.23	63.9-91.5	60- 96
50	26	11	78.2	7.2	1.44	71.0-85.4	64- 90

*σ = standard deviation of the mean.
σm = standard error of the mean.

As the intensity is raised to 15 db or more above the threshold for Test No. 9, the curves diverge significantly. The means for patients with mixed-nerve loss are *significantly* lower than for those with conductive-mixed loss at all intensities 15 db or more above the threshold for Test No. 9. The level of confidence that this is a real difference is between .01 and .001, or better, except at 20 db above the threshold for Test No. 9, where it is .02. (The fraction .01 indicates that the probability is not more than one in a hundred that the result found could have occurred simply because of ran-

dom differences. The fraction .001 indicates that the probability is only one in a thousand, and so on). The conductive-mixed curve diverges *significantly* from the pure-conductive curve at 25 db above the threshold for Test No. 9. From 25 to 50 db above the threshold for Test No. 9, the means for conductive-mixed loss are *significantly* lower than those for pure-conductive loss. (The level of confidence is better than .001, with the exceptions that at 30 db above the threshold for Test No. 9 the level of confidence lies between .05 and .02 and no significant difference was found at 55 db above the threshold for Test No. 9.) At levels well above the threshold for Test No. 9, therefore, the group of patients with nerve involvement gives a significantly inferior performance on the Rush Hughes recording, and the greater the nerve involvement, the nearer threshold the inferior performance shows up. The data thus confirm the theory of Walsh and Silverman that performance on the PB test is correlated with clinical diagnosis.

Maximum Articulation Scores

In clinical practice, we have considered it most useful to give the PB test at an intensity level where the patient will be able to give his maximum performance, in other words to attempt to measure the highest point on his articulation function. The question arises of how far above the threshold for Test No. 9 the intensity should be set in order to measure the maximum PB articulation score, or "PB-Max". The articulation functions never reach 100 per cent, and their upper portions show a very gradual but significant improvement in score with increasing intensity, rather than a well-defined plateau. The means (for patients with pure-conductive loss) at 50 and 55 db above the threshold for Test No. 9 are *significantly* higher than the mean at 40 db above. (The level of confidence for this difference is better than .001). It is clear that to measure the maximum articulation score, the test should be given at least 40 db above the threshold for Test No. 9. It is not always practical, however, to give the test at such a high level, particularly to patients with considerable

hearing loss, as the required level may be above the threshold of discomfort; but if the test is given at lower levels, a correction can be applied to the measured score in order to obtain a better estimate of the true maximum value. Table IV lists the number of percentage points that should be added to the observed score according to the level at which the test is actually administered. These corrections are based on the shape of the average articulation function for all of the patients, including those with some nerve deafness as well as those with purely conductive deafness.

TABLE IV.

CORRECTION FOR RUSH HUGHES "PB-MAX" SCORES OBTAINED BELOW THE TRUE MAXIMUM.

Level Decibels Above Threshold for Test No. 9	Correction Percentage Points; Add to Score
25	14
30	8
35	4
40	1
45	0

The differences between the mean "PB-Max" scores for the three diagnostic groups were determined by averaging for each group separately all of the tests given from 40 to 55 db above the threshold for Test No. 9. The results are shown in Table V. The differences between the mean "PB-Max" scores are *significant* at a level of confidence better than .001.

TABLE V.

MAXIMUM PB ARTICULATION SCORES.

Diagnosis	No. of Tests	Mean Score (Per Cent Correct)	σ	Mean Score $\pm\sigma$
Pure Conductive	1036	89.7	6.2	83.5 to 95.9
Conductive Mixed	421	86.0	6.8	79.2 to 92.8
Mixed Nerve	140	75.8	10.8	65.0 to 86.6

Our test differentiates most clearly between pure-conductive and mixed-nerve loss. There is very little overlap between the ranges of plus and minus one standard deviation around the means for these two groups. The bottom of this range for pure conductive loss is 83.5 per cent, and the top for mixed nerve is 86.6 per cent. Eighty-five per cent, the center of this small area of overlap, may be taken as a practical dividing line between scores showing good discrimination and those showing impaired discrimination.

The percentages of the total number of "PB-Max" tests which fall into various score ranges is useful in the interpretation of individual tests. This information is given in Table VI.

TABLE VI.

DISTRIBUTION OF MAXIMUM ARTICULATION SCORES.

"PB-Max" Score (Per Cent Correct)	PURE CONDUCTIVE		CONDUCTIVE-MIXED		MIXED-NERVE	
	No. of Tests	Per Cent of Tests	No. of Tests	Per Cent of Tests	No. of Tests	Per Cent of Tests
90 to 100	616	59.5	135	32.1	5	3.6
80 to 88	359	34.6	221	52.5	53	37.8
70 to 78	59	5.7	60	14.2	56	40.0
68 and below	2	0.2	5	1.2	26	18.6
TOTAL	1036	100.0	421	100.0	140	100.0

Since the mean score for pure-conductive loss is just under 90 per cent, it appears that a score of 90 per cent or higher on the Rush Hughes test is excellent. Seventy-eight per cent, on the other hand, is a poor score, strongly indicating nerve involvement. Scores from 80 per cent to 88 per cent are inconclusive, since patients in all three groups score in this range.

Reliability

There is considerable dispersion of scores around the means in all parts of the articulation function. The standard devia-

tions are shown in Tables I, II, and III. Even at the highest input levels where the spread is smallest, the standard deviations do not become much lower than 6 per cent. The standard deviations for the conductive-mixed and for the mixed-nerve patients are even greater than those for the pure conductive patients.

The reliability of the "PB-Max" has been examined and discussed at some length in previous publications.[7,8,9] The principal additional information contributed by Tables I, II, and III is that the standard deviation for scores obtained on the steeper part of the articulation function is approximately double the standard deviation of scores on or near the plateau.

Because of the rather wide dispersion of the scores, measured by the standard deviation, we feel that if any diagnostic significance is to be attached to the "PB-Max" score it is important to employ at least 2 PB lists. The mean score for the two lists is appreciably more reliable than either score alone. The number of PB lists employed in a given session cannot be increased indefinitely, however, for the test requires several minutes and the constant loudness of the PB words gives the patient a greater impression of monotony than do the changes of intensity in the threshold test. The patient's attention may become a critical factor; moreover, the purpose of the threshold test is more obvious to the average patient than the purpose of the articulation test even if the latter is carefully explained in advance.

Another factor which may have contributed to the dispersion of scores on the articulation tests is vocabulary. Some of the words employed in the PB lists were not familiar or only slightly familiar to many of our patients. The question of familiarity seems to have been much more important in the PB tests than in Auditory Test No. 9. The latter is composed of words of two syllables that were originally chosen because of familiarity as well as their relative uniform intelligibility. We suggest that in the tests of discrimination the familiarity of test items be given careful consideration.

Relative Difficulty of Rush Hughes PB Lists

Three studies have been carried out to measure differences in difficulty between the eight Rush Hughes PB lists. The first consisted of grouping the "PB-Max" scores of our pure conductive cases according to list and comparing the mean score for every list with that of every other list by means of the t-test. In the second study, the same procedure was followed using the "PB-Max" scores of the conductive-mixed group. The third study was a planned comparison of lists carried out on consecutive patients as they came to McMillan Hospital for testing, without selecting the subjects on the basis of diagnosis. In this study the lists were paired systematically and the maximum articulation score was determined with each pair for each of twenty hard-of-hearing ears. The intensity of the two experimental lists was the same for each ear tested, and the sequence of the two lists was varied from patient to patient to counterbalance fatigue and learning effects. The method of pairing was as follows: Lists 6 and 12 were used as reference lists; List 6 was first paired with Lists 5, 7 and 8; then List 12 was paired with Lists 9, 10 and 11; and finally Lists 6 and 12 were compared. In this way, the eight lists could be arranged in order of difficulty on the basis of average differences in score.

The order of difficulty of lists, from easiest to hardest, as indicated by the three studies, is as follows:

All Conductive cases	11, 12, 10, 7, 5, 9, 6, 8
All Conductive-Mixed cases	12, 11, 10, 7, 9, 6, 5, 8
Planned Comparison series	12, 11, 9, 7, 5, 10, 6, 8

In all three studies, Lists 11 and 12 were found to be easiest, and List 8 hardest. The statistical evidence of this was more striking in the conductive than in the conductive-mixed study. For patients with pure conductive loss, List 8 was significantly harder (by the t-test, level of confidence .05 or better) than every other list, while for the conductive-mixed group it was significantly harder than four other lists. List 11 was significantly easier than six other lists in the conductive

study, but it was significantly easier than only three lists in the conductive-mixed study. Perhaps the reason for the more uniform difficulty of the lists for the conductive-mixed group is that patients with some nerve involvement make lower scores. They, therefore, make mistakes not only on the five or six most difficult items, but also on the ten or twelve most difficult. As more items become critical, the lists apparently become more nearly equivalent.

List 11 is easier than List 12 (for the conductive group) probably because it contains no extremely difficult items, while List 12 includes the words "frill" and "greet" which are very frequently missed. More patients are, therefore, able to make 100 per cent on List 11. Of our group of 1036 "PB-Max" scores on pure conductive cases, there were 27 perfect scores, 14 of which were made on List 11.

A detailed study of the significance of the differences in difficulty among the five other lists in the three studies has led us to the general conclusion that Lists 5, 6, 7, 9 and 10 are of practically equal difficulty. Such significant differences as appeared in one study were not confirmed in the other two. List 8 is, however, *significantly* harder and Lists 11 and 12 are *significantly* easier than the other five. The differences in average "PB-Max" scores for the different lists are of the order of only 2 per cent.

If the Rush Hughes recordings of Lists 8, 11 and 12 are to be employed for determining the maximum articulation scores of patients with conductive or mixed hearing losses, the following corrections should be applied:

List 8:	add 2 percentage points to the apparent "PB-Max" score
Lists 11 and 12:	subtract 2 points from the apparent "PB-Max" score.

These corrections are to be applied in addition to the correction for intensity given in Table IV. The corrections are small, but they are important because the differences between the mean "PB-Max" scores of the diagnostic groups are not large.

DISCUSSION.

We formerly believed[2,3] that the articulation function for patients with pure conductive hearing loss was a simple displacement on the intensity scale of the normal articulation function; however, the curve which we have now constructed for the conductively deaf differs from the old "normal" curve in three respects. The score at the level of the threshold for Auditory Test No. 9 is about 20 per cent, not 8 per cent; the curve is less steep; and it never reaches 100 per cent. It continues to rise gradually and significantly at levels as much as 50 db above the threshold for Test No. 9, but it never gets much higher than 90 per cent. Just how significant these differences from normal are we cannot tell until data are available on a large group of normal but untrained listeners.

The data show several characteristics of the Rush Hughes recording which might be improved upon in any new recordings to be made in the future. The wide range of difficulty among individual test items is probably not ideal. It is true that, in a clinical test, the easiest items should be easy enough to make the test practical for testing patients with very poor discrimination, and the most difficult items should be hard enough to make the test sensitive to small differences in discriminatory ability found among normals and among patients with pure conductive hearing loss; but the most difficult items must not be so hard that the test becomes unrealistic. The range of difficulty in the Rush Hughes recording may be too wide; also, it would be a practical advantage in a clinical test if a definite plateau were reached at 35 db above the threshold for spondaic words. It would then more often be possible to measure the maximum articulation score directly without using a correction.

There is evidence that the distribution of difficulty among items in the Rush Hughes recordings is uneven. We have performed an item analysis of the errors made by conductive-deaf patients on three of the eight individual lists. The analysis was confined to the tests given 40 to 55 db above the threshold for Test No. 9. It was obvious that certain words

such as "flip", "spice" and "deuce" in List No. 8 were particularly difficult and that the other lists contained fewer or no items of comparable difficulty.

There is an irregularity at the 20 db level in the shape of the average articulation functions for all lists (see Fig. 1). This depression is significant for the pure-conductive and for the conductive-mixed groups. We believe that this irregularity is a spurious characteristic of the Rush Hughes recordings.

We hope that our experience with the Rush Hughes recordings will prove valuable to those who attempt to produce better recorded versions of the PB lists in the future.

SUMMARY.

1. A set of recordings by Rush Hughes of eight "phonetically balanced" word lists has been used for five years at Central Institute for the Deaf and in the Hofheimer Audiology Laboratory (McMillan Hospital) to test the auditory discrimination of candidates for the fenestration operation. From the data which have been collected, composite articulation functions for the Rush Hughes recordings, based on the threshold for Auditory Test No. 9, have been constructed for three groups of patients, diagnosed respectively as follows: (a) pure conductive loss; (b) mixed loss, predominantly conductive; and (c) mixed loss, predominantly nerve. In averaging the articulation scores of different patients, each patient's threshold for Auditory Test No. 9 was taken as the reference point.

2. Comparisons by the t-test of the mean scores of the three clinical groups at levels from the threshold for Auditory Test No. 9 to 55 db above it show that, at levels 25 db and higher above the threshold for Auditory Test No. 9, performance on the Rush Hughes recordings of PB lists is correlated with clinical diagnosis, as suggested by Walsh and Silverman in 1946.

3. Since the articulation functions are still rising gradually but significantly 40 and 45 db above the threshold for Test No. 9, the intensity chosen for measurement of a patient's maximum PB articulation score should be as far above his threshold for Auditory Test No. 9 as is practical, if his hearing loss is wholly or partially conductive.

4. The articulation functions indicate that the range of difficulty among individual test items in the Rush Hughes recordings is very wide and that the distribution of difficulty is uneven.

5. The reliability of the Rush Hughes test is not very high. At levels 40 to 55 db above the threshold for Auditory Test No. 9, the standard deviations around the mean scores never become much lower than 6 per cent.

6. Comparisons of the eight Rush Hughes PB lists show that List 8 is significantly hard, Lists 11 and 12 are significantly easy, and Lists 5, 6, 7, 9 and 10 fairly well equated in difficulty. Correction factors to be used with Lists 8, 11 and 12 are given.

7. Our experience with the Rush Hughes recording points out certain problems which must be considered when new recordings of PB lists are made.

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THE TONGUE AS A FOCAL INFECTION.

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In this age of exact scientific method, bacteriologists, pathologists and internists are extremely skeptical of proof obtained by clinical evidence alone. The doctrine of focal infection has not been completely proved by scientific methods and, therefore, has become quite controversial. As a matter of fact, judging by recent publications, its pendulum has swung pretty low, nor is this paper to hold up the tide against it. Verhoef[1] states, "Belief in focal infection must be taken like religion — on faith." Slocum *et al.*,[2] of the Mayo Clinic, evaluate the concept of focal infection on clinical observation alone. In much the same way, I shall cite a few cases where the therapeutic response to the eradication of the focus of infection seems to justify this doctrine as a working hypothesis, if nothing more. Woods[3] states: "It is an established procedure with the ophthalmologist to search for foci of infection in the study of uveal tract diseases." Many other diseases of the eye and its appendages may also be cured by eliminating foci of infection. Thus in another article[4] written in 1932, I illustrated that dacryocystitis may be relieved by eliminating foci of infection. A very convincing clinical proof of focal infection is the following case:

Case 1: Female, aged 30, had a peritonsillar abscess in 1919 which was opened and drained. She was then advised to have her tonsils extirpated, but she refused. Four years later she had a fulminating optic neuritis in one eye with hemorrhages and exudates all around the papilla, which was not elevated. Upon examination of the upper respiratory tract, it was found that the tonsils were infected and two gold-capped teeth were present. X-ray of the teeth revealed no infection. The tonsils were removed and 48 hours later the eye cleared, and the vision returned to normal.

Four years later she had a similar attack in the same eye. She was examined thoroughly by an internist, had X-rays taken of all parts of the

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body, and was subjected to all other possible means of investigation. All tests were found to be absolutely negative. Three weeks were consumed in the examinations, and the patient did not get any better. It was only after an X-ray of the teeth that an abscess was found, which was eliminated by extraction. The eye cleared of the acute infection within 48 hours. It did not return to normal, however, being left with a partial secondary atrophy of the optic nerve.

Two years later she had another attack in the same eye. She was then edentulous; tonsils were extirpated with no residual tissues visible, and sinuses were clear on transillumination and X-ray. She volunteered the information that she had some gynecological disorder. She was sent to a gynecologist, who reported that she had a definite chronic endocervicitis and recommended cauterization. This having been done, in 48 hours the eye again cleared to the status preceding the last episode.

Against this clinical evidence grave objections may be raised, chiefly that patients do get well spontaneously and that some patients who had their tonsils removed, teeth extracted and perhaps sinuses punctured obtain no results, or are possibly made worse. With regard to the former point, Woods[3] claims that the clinical evidence for recovery on removal of foci of infection is so formidable that it is difficult to dismiss it lightly on the ground that it is only suggestive. With regard to the latter point, Slocum *et al.*[2] demonstrated that where the malady is irreversible or irreparable, removal of foci will be of no therapeutic benefit. On the other hand, can we honestly say that we have eradicated all foci of infection when we have eliminated a few obvious ones? Lederer[5] claims that the phrase "ear, nose and throat essentially negative" does not mean anything unless we have used all means at our disposal for examination, such as the laryngeal mirror to investigate the lingual tonsils, the nasopharyngoscope or a postnasal mirror for nasopharynx, transillumination and X-ray of the sinuses. Otherwise, we leave out many possible sources of infection.

Davis[6] reported cases which he had miraculously relieved of pain and remote disease by cauterizing some infected follicles of the lingual tonsils or lateral bands of the pharynx or follicles anywhere in the nasopharynx. He concludes by saying: "*1.* Tonsils do not constitute the only foci of infection in the pharunx. *2.* Other foci are lingual tonsils, adenoids and lymphoid tissue on the posterior pharyngeal wall."

There still remains another possible focus of infection in the oral cavity to which very little attention has been paid, and that is the tongue—particularly the coated tongue. Gray[7] states that the tongue presents a depression, the foramen cecum from which a shallow groove, the sulcus terminalis, runs lateralward and forward on either side to the margin of the tongue. The part of the dorsum of the tongue in front of this groove, forming about two-thirds of its surface, looks upward, and is rough and covered with papillae; the posterior third looks backward and is smoother, and contains numerous muciparous glands and a large number of lymph follicles, which together constitute what is sometimes termed the lingual tonsil. Elfman[8] claims that the lingual tonsils are compensatory to faucial tonsils and are similar in structure to them. They are subject to the same affections and may act as focal infection in the same manner as the faucial tonsils do. A striking example is the following case:

Case 2: C. E., female, aged 23 years, had a history of general glandular enlargement, duration of one year, accompanied by low-grade fever and malaise. All tests were made and proved negative, including complete hematological and serological tests and biopsy of one gland. Examination of the upper respiratory tract revealed enlargement of the lingual tonsils with follicles all over the posterior part of the tongue. These follicles from time to time became acutely inflamed. A higher elevation of temperature occurred with the acute inflammation. She was anesthetized, and the lingual tonsils were removed along with the surrounding lymphoid follicles. She made an uneventful recovery, swelling of all glands subsided, temperature became normal, and she has remained well ever since, a period of 12 years.

The papillae covering the anterior two-thirds of the tongue are minute surface eminences. They consist of a layer of connective tissue covered with epithelium. The filiform papillae are the most numerous, occupying the general area anterior to the large circumvallate papillae, which form the inverted V-shaped terminal sulcus at the base of the tongue.

Crohn and Drosd[9] state that each filiform papilla is surrounded by projections, the "secondary" papillae which protrude like flexible spines or spikes, covered by stratified squamous epithelium.

Henning[10] found the filiform papillae to be plump, round hillocks with transparent epithelium. Most characteristic were

the secondary papillae which jutted out from the filiform papillae like blades of grass with serrations. When the tongue was coated the papillae were long, thick, firm and tense, and were surrounded by leucocytes, bacteria and particles of debris.

Bloomfield[11] demonstrated that bacteria grow normally not only on mucous membrane but also in their superficial layers, in crevices of the superficial layers and between them, and in orifices of small glands.

The coating of the tongue according to Boas[12] is ideal for the development of an offensive odor of putrefaction and calls it "fetor ex lingua." While fetor ex ore can be produced by many disorders, such as gastrointestinal diseases, lung abscesses, bad teeth and their surrounding tissues, infected sinuses, malignancies in the throat and mouth, there is sometimes an unique odor of putrefaction without any demonstrable malady; however, the tongue is usually coated in these cases and washings from it emanate the same characteristic odor. That this odor can be produced by the tongue alone, I have proved in an earlier paper[13] written in 1932, which was demonstrated with the following case:

Case 3: B. L., child, female, 12 years old, had been suffering from bad breath for several years. Tonsils and adenoids were removed with no beneficial effect. She was surveyed on two different occasions in two leading hospitals and nothing abnormal could be found, except a slight postnasal discharge, for which she was treated. The discharge when removed from the nasopharynx proved to be odorless. There was no bad taste complaint, the appetite was good, and the weight was normal. In spite of the fact that the gastrointestinal tract was found to function properly, her tongue was always coated with a white fur. Washings from it had the identical odor which one always detected on her breath.

The fetor ex lingua was not eliminated until we started the use of antibiotics (aureomycin). Cleansing of the tongue alone produced only temporary relief of the fetor.

Lately, in searching for foci of infection of the upper respiratory tract, I came across patients with fetor ex lingua. Upon eliminating the fetor, the remote malady for which a search for a focus was made, also began to resolve without any other infection presenting itself. I then made it a rule to examine the whole tongue routinely. When coating is present

and washings have a characteristic odor of putrefaction, the aim is to clear up that condition first. Many times the patient got well without further treatment, and without finding another focus of infection. Crowe's[14] statement regarding "the theoretical explanation on which is based the otolaryngological treatment and preventive measures for recurring upper respiratory infections and their complications" can be applied to the tongue's acting as a focus of infection. Pyogenic bacteria grow on the coated tongue just as they would in a test tube. These cannot initiate infection; but when the tongue becomes infected with a virus, which is an intracellular parasite, it opens a pathway of infection. The infection may thus be carried through the blood stream and act as a focus of infection for remote diseases. Several cases of iritis cleared up by simple cleansing of the coated tongue with aureomycin in suspension (50 mg. to one dram of normal saline) and aureomycin internally, as exemplified by the following case:

Case 4: A. C., male age 20, came under my observation June, 1939. Diagnosis was iritis. Conservative treatment made no impression. Infected tonsillar tags were found and cauterized, and the eye cleared up quickly. May 7, 1946, he had a similar attack of iritis. Empyema of both antra was present and cleared up with washings. The eye cleared up very quickly with simple treatment of atropine. Dec. 3, 1950, he had another attack of iritis. This time no focus could be found. Milk injections were given with no appreciable effect. He had a fetor ex ore with the tongue coated and washings elicited a foul odor of putrefaction. This was cleaned and aureomycin given internally and the eye cleared up quickly.

Another case of iritis also of recurrent nature was given aureomycin but with very little effect. The tongue was found to be infected and when this was cleared up the eye got well.

One case of optic neuritis was treated with cortisone, aureomycin, and milk injections with no effect. She had complete physical and laboratory tests taken, including a spinal tap, with all negative findings. She had a fetor ex ore which was found actually to be fetor ex lingua. This was cleaned up with aureomycin suspension locally, and the clearing of the eye followed very quickly.

Two cases of dacryocystitis, as mentioned above, have been cleared up with the elimination of foci of infection on the

tongue. One case was of a purulent nature which had to be incised externally, and the other had simple epiphora.

Ten cases with Ménière's syndrome were also cleared up by the hygiene of the tongue. They were all of a toxic nature, but no focus could be found other than the coating of the tongue with putrefactive material. These may be exemplified by the following case:

Case 5: A. D., male, age 60, was treated for catarrhal deafness with the Eustachian tube obstructed. He was doing fairly well when he began to have evidence of Ménière's syndrome. I diagnosed this case as a toxic labyrinthitis, but I could not find a focus of infection. He was given two courses of aureomycin, and he improved somewhat. When medication was stopped his symptoms recurred, and he became a chronic vertiginous patient. Upon the slightest provocation he would have vertigo of variable degrees and duration. Further checking on him elicited a coated tongue, washings from which emanated the characteristic odor of putrefaction. Treatment of the tongue was then instituted, the patient improved very rapidly.

Since then nine cases of Ménière's syndrome were seen in patients with more or less of a catarrhal deafness. The impairment of hearing was not affected by the attack nor was tinnitus complained of. They all had coated tongues with odor of putrefaction. When treated as outlined above, good results were obtained in all of them. Three patients came soon after the first attack. Their tongues were coated only a short while and responded to local treatment alone.

Conclusion: The coated tongue may act as a focus of infection and sometimes be the only focus for a varied number of diseases. Every coated tongue should have washings examined, particularly as to odor. If putrefaction is present, the tongue should be cleaned thoroughly with antibiotics and antibiotics given internally. When putrefaction has been existing for a long time, treatment should be prolonged, since recurrence of the odor and the malady are likely to reappear.

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INTRANASAL MENINGOCELE.

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Meningoceles occur very rarely. At the Birch-Hirschfield Clinic[3] not a single case was found in 200,000 examinations. McGillicuddy[4] found nine authentic cases in the literature up to 1942, all but two of which terminated by meningitis. To these he added a case of his own which survived surgery and postoperative meningitis, but is still suffering from spinal rhinorrhea. In 1947, New and Devine[6] reported that only six cases of meningocele had been discovered at the Mayo Clinic during the past 27 years. Five were frontal; the sixth, an intranasal case, was complicated by spinal rhinorrhea.

Meningoceles result from the herniation of pia and arachnoid through a dehiscence in the skull. Frontal meningoceles usually present at the root of the nose as a soft, compressible, symptomless tumor which may pulsate.

Intranasal meningoceles may result from herniation through the sphenoid or ethmoid. Stuart,[5] of the Montreal Neurologic Institute, found a meningocele which extended into the nasopharynx. In a case reported by Sedan[3] the tumor filled the ethmoid area and presented near the inner canthus of the eye. O'Brien's[2] case was even more remarkable in that a cystic middle turbinate was found to contain a meningocele.

More commonly, however, intranasal meningoceles occur as a result of a dehiscence in the lamina cribrosa. In such cases the hernial sac consisting of pia and arachnoid and filled with spinal fluid forces its way through a rent in the dura into the

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interior of the nose. Here, attached to the septum, it presents as a grayish, semitranslucent, sessile tumor which is covered by septal mucosa.

Intranasal meningoceles have been confused with polypi. This error should not occur if one remembers that polypi in infants are exceedingly rare, that they are pedunculated and originate near the ostia of the sinuses on the lateral side of the nose.

Other criteria have been suggested to aid in the differential diagnosis. Compression of the jugular veins may produce a light reflex and distension of the meningocele. Lumbar puncture and drainage may decrease its size and tension. X-rays have demonstrated the defect in the cribriform plate.

The treatment of intranasal meningoceles has been far from successful. Only three cases reported in the literature have ended in recovery. Biopsy and aspiration have resulted in a fatal meningitis in many cases.

The treatment of choice is ligation of the pedicle either via the radical frontoethmoid approach or by craniotomy.

CASE REPORTS.

The first case is that of a three-year-old white male who has a symptomless, soft, compressible tumor mass at the root of the nose, 2 cm. in diameter.

The diagnosis of frontal meningocele was made, but since no immediate treatment was advised the patient was not seen again.

The second case is that of a white male infant, 23 months of age. At about nine months of age he developed a constant discharge and obstruction of the left nostril.

He had a nasal operation in January, 1950, an adenotonsillectomy in March, and another nasal operation in April.

On July 17, 1950, when first seen in consultation the patient had an obvious nasal obstruction on the left. The nostril was occluded by a pale, soft tumor mass covered by glary mucus. Adenoid tissue partially occluded the left choana.

Following a course of X-ray therapy, the nose was dry and the child breathed normally for several months.

When next seen, Nov. 27, 1950, the nasopharyngeal lymphoid tissue was absent, but his left nostril was still obstructed.

On the suspicion that we were dealing here with an intranasal meningocele, he was examined under general anesthesia. After cleansing the left nostril of mucus, the tumor was found to be continuous with the mucosa of the septum and free from any lateral attachment. The nostril was carefully packed with adrenalin gauze so as not to traumatize the tumor, and the packing left in place for five minutes. When it was removed the tumor had disappeared, but the sac could be seen filling the olfactory groove. It gradually expanded while under observation.

Fig. 1. The lateral X-ray of the anterior part of the skull revealed the dehiscence in the cribriform plate between the arrows.

On the basis of these findings, the diagnosis of intranasal meningocele appeared to be warranted. Accordingly, the patient was referred to Dr. William Lipscomb for craniotomy. Dr. Lipscomb's report follows:

The diagnosis of intranasal meningocele was made by Dr. Beyer and Dr. Blair, so when the patient was referred for treatment he was admitted to Children's Hospital where X-rays of the skull were taken which revealed, in a lateral projection (see Fig. 1), a dehiscence in the cribriform plate, anteriorly. In an anterior tangential X-ray view (see Fig. 2)

Fig. 2. The anterior tangential X-ray of the skull revealed the marked enlargement of the left naris.

of the skull the left naris was much larger than the right. On Nov. 29, 1950, under general inhalation, intratracheal ether anesthesia combined with a field block of 1 per cent procaine containing 1:200,000 epinephrine, a left transfrontal craniotomy was done. An extradural approach was made toward the sphenoidal ridge where a long dural incision was made so that the left frontal lobe could be elevated and dislocated posteriorly. Near the anterior tip of the left frontal lobe on the medial, inferior aspect a small stalk (approximately 3 mm. in diameter) of arachnoid-like tissue was encountered which was binding the arachnoid over the tip of the medial, inferior aspect of the left frontal lobe near the longitudinal fissure to a depression in the olfactory groove of the anterior fossa. The stalk was doubly ligated with No. 50 cotton sutures and sectioned between the ligatures. With the bayonet forceps the dehiscence in the cribriform plate could then be easily palpated. It measured approximately 1 cm. in length and 3 mm. in its widest dimension. The dura was scarified for approximately 1.5 cm. surrounding the sectioned stalk over the cribriform plate and a free pericranial graft was taken and placed to cover the sectioned end of the arachnoidal stalk and the bony dehiscence. The area was suctioned dry in order to promote adherence of the graft. The brain was then allowed to fall into normal position and the wound closed in layers. A Penrose drain was left in the wound two days and 10,000 units penicillin were injected into the wound after closure. In order to get the brain to tampon the pericranial graft over the bony and dural defect an effort was made to keep the patient lying on the right side with the head in right oblique position. In a 23-month-old child such an effort was only partially successful.

On Jan. 19, 1951 (51 days postoperative), the patient was brought to the office by the parents who reported he was "nervous and didn't sleep well, but that he had had no nasal symptoms." On examination, however, he was alert, agile, interested and happy. Neurological examination and an external examination of the nose revealed the little fellow to have no nasal drainage and to have no obstruction to the passage of air.

CONCLUSIONS.

1. Meningoceles occur very rarely.
2. Intranasal meningoceles may be confused with polypi.
3. Aspiration and biopsy are dangerous procedures.
4. X-ray studies proved to be a valuable diagnostic adjunct.
5. Intracranial ligation of the pedicle appears to be the treatment of choice.

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ACUTE INFECTIOUS ATELECTASIS SIMULATING BRONCHOPNEUMONIA IN INFANTS.

SYMPTOMATOLOGY AND TREATMENT.

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The death rate from pneumonia in children under the age of one year is surprisingly high in this country, 13 to 17 per cent, and every effort should be made to reduce it. Serious congenital malformations may play some part in the higher mortality of the younger infants, but in the majority of fatal cases there seems to be no other explanation except delay in the institution of treatment or underdosage of the drug employed prior to admission to the hospital. On examining the autopsy records of the Pediatric Department of Sundby Hospital, it was striking to find that the rather frequent demonstration of atelectasis postmortem had not been suspected clinically; nor has sufficient attention been given to this aspect of the problem by other authors.

No doubt the main reason that the morbid condition, which in Sundby Hospital we have called "acute infectious atelectasis," has not yet been established clinically is because X-ray examination is often not considered justifiable in these markedly exhausted children, who must be kept at rest as much as possible and treated only with oxygen and chemotherapy. For this reason, the chance of recognizing the true nature of the lesion is lost.

As an illustration of the clinical picture of *acute infectious atelectasis in infants* it would be appropriate to summarize here two characteristic case histories of children admitted to

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this department in one month, both under the diagnosis of pneumonia—one of them almost moribund at the time of admission:

Case 1: No. 107/50. Girl, three months old. Admitted Feb. 3, 1950, and discharged on March 20, 1950.

The child was born two to three months before term. Bottle-fed. One week before admission showed restlessness, dyspnea, vomiting and a few thin stools.

Physical Examination: On admission, the child was markedly exhausted, grayish, breathing very rapidly (respiration rate 70 per minute). Dilatation of the nostrils and pronounced inspiratory retraction of the suprasternal notch and epigastrium, but no stridor.

Auscultation of the Lungs: Semimoist, subcrepitant rales heard over the anterior and posterior surfaces. Temperature, 36.8° C.

Treatment: Sulfathiazole, penicillin, streptomycin and oxygen.

As atelectasis was suspected, an X-ray picture was taken of the child in the ward with a portable X-ray apparatus. It showed atelectasis of the right upper lobe (see Fig 1A). On the day after admission, bronchoscopy was performed with the Negus baby bronchoscope (Dr. Riskaer). A very abundant tenacious greenish-yellow secretion was found in the trachea and both main bronchi, especially in the right. The mucous membrane was swollen, again especially on the right side, where the lumen was distinctly narrowed. The secretion was aspirated (cultures gave growth of pneumococcus type 33 and B. coli), and oxygen was administered. After this, the respiration improved considerably.

Eight hours later the condition of the child again deteriorated, and death seemed imminent. The respiratory rate was about 100 per minute in spite of stimulation with lobeline and oxedrin.

Bronchoscopy was repeated, with aspiration of a considerable amount of tenacious secretion, though distinctly less than before. On introduction of the bronchoscope, which was easy enough, the respiration stopped, but was soon re-established by administration of oxygen through the bronchoscope. For the next several days the condition of the child improved strikingly. On discharge, the child was perfectly well. The Roentgenographic findings are reproduced in Fig. 1.

Case 2: No. 182/50. Girl, two months old, breast-fed. Admitted March 4, 1950, and discharged March 15, 1950. The child was born at term by spontaneous delivery. Previously well. Four days before admission she developed coryza with cough. The temperature did not exceed 37.5° C. On the day before admission the child was distinctly dyspneic, crying and restless. She was treated at home with steam and sulfathiazole—without any improvement.

Physical Examination: On admission the child was lively and not particularly distressed. Respiratory rate 40 per minute, with distinct inspiratory retraction of the epigastrium.

Auscultation of the Lungs: Breath sounds weak over the right posterior surface, and a few subcrepitant rales. Temperature 37.2° C. *Clinical Diagnosis:* Bronchopneumonia.

Fig. 1-A. Fig. 1-B. Fig. 1-C.

Fig. 1-A, 1-B, 1-C. Case 1. Girl, three months old. (A) Atelectasis of the right upper lobe (Feb. 4). (B) Three days after bronchoscopy with aspiration. Atelectasis almost subsided (Feb. 7). (C) Atelectasis cured (Feb. 11).

Fig. 2-A. Fig. 2-B. Fig. 2-C.

Fig. 2-A, 2-B, 2-C. Case 2. Girl, two months old. (A) Atelectasis of the right upper lobe (March 5). (B) Twenty-four hours after bronchoscopy and aspiration (March 6). (C) Three days after bronchoscopy with aspiration. Atelectasis cured (March 8).

Treatment: Penicillin, streptomycin, steam and oxygen. The following morning (March 5) there was an obvious deterioration in the condition of the child. X-ray examination of the lungs (performed in the ward) showed atelectasis of the right superior lobe (see Fig. 2A). Bronchoscopy was then performed. On aspiration, fresh amounts of secretion appeared continuously into the bronchus, so that 10 to 12 cc. in all were aspirated (see Fig. 3). The bronchoscopy was tolerated well. Cultures from the aspirated secretion yielded growth of Gram positive cocci (staphylococcus aureus and enterococci) and Gram positive rods.

Fig. 3. Case 2. Secretion withdrawn at the bronchoscopy.

Five hours later the bronchoscopy was repeated and 3 to 4 cc. of very tenacious secretion were aspirated from the right main bronchus.

After this, the condition of the child improved considerably, and the respiratory rate fell from 80 to 52 per minute.

Three hours later bronchoscopy was carried out for a third time, but there was only a scanty amount of secretion present.

Twelve hours later (March 6) the child appeared to be perfectly well and was discharged after 11 days in the hospital.

The Roentgenographic findings are reproduced in Fig. 2.

The admission of these two patients within one month might be taken to indicate that *acute infectious atelectasis* is not altogether infrequent in infancy.

In order to look into the frequency of this lesion, therefore, we have gone through the autopsy records of those children who died under the diagnoses of pneumonia, bronchitis or laryngotracheobronchitis fulminans in the pediatric department of Sundby Hospital and Blegdam Hospital (Epidemic Diseases)* for the last five years.

The frequency of atelectasis is shown in Table 1.

TABLE 1.

Atelectasis demonstrated by autopsy in 178 children who died under the diagnosis of pneumonia, bronchitis or laryngotracheobronchitis in 1945-49.

	Sundby Hospital				Blegdam Hospital			
	+ Atelectasis				+ Atelectasis			
Age	Focal	Extensive	Total	No Atelectasis	Focal	Extensive	Total	No Atelectasis
3 weeks-3 mons.	6	3	9	18	4	7	11	10
3- 6 mons.	2	4	6	2	10	4	14	18
6-12 mons	0	5	5	4	12	7	19	27
1- 2 yrs.	0	1	1	2	7	2	9	12
2-yrs.	0	0	0	2	1	1	2	7
Total	8	13	21* (43%)	28 (57%)	34	21	55† (43%)	74 (57%)

*In three of these cases the clinical diagnosis was laryngotracheobronchitis.
†In two of these cases the clinical diagnosis was laryngotracheobronchitis.

The figures given in Table 1 are the absolute minimal figures, as in a number of these cases no histological examination was made, and atelectasis cannot always be distinguished from pneumonia by macroscopic examination alone.

In this material the diagnosis of atelectasis was made clinically only in *one case* — that of a boy, three months old, who

*We are greatly obliged to Prof. H. C. A. Lassen, M.D., Chief of Blegdam Hospital, for permission to make use of the case records.

was admitted to Blegdam Hospital for whooping cough. The diagnosis was made Roentgenographically, and bronchoscopy was not performed. The child was treated with penicillin and sulfathiazole, but died nine days after admission—complete atelectasis of the right lung developed gradually (see Figs. 4A and 4B).

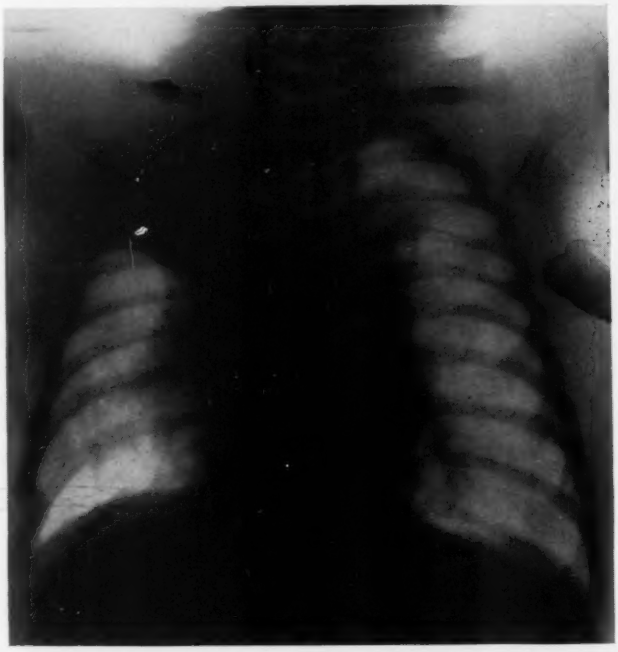

Fig. 4-A. Boy, three months old. Atelectasis of right upper lobe (Sept. 26, 1948).

Among the 129 patients in the autopsy material from Blegdam Hospital, 1945-1949 (*cf.* Table 1), X-ray examination was performed only on two other patients (two boys, five and eight months old). In each of these cases the X-ray diagnosis was pneumonia, but autopsy (carried out one and two days after the X-ray) revealed the presence of atelectasis in both,

and no pneumonia. At the X-ray examination no lateral picture was taken, and perhaps this may explain the erroneous X-ray diagnosis.

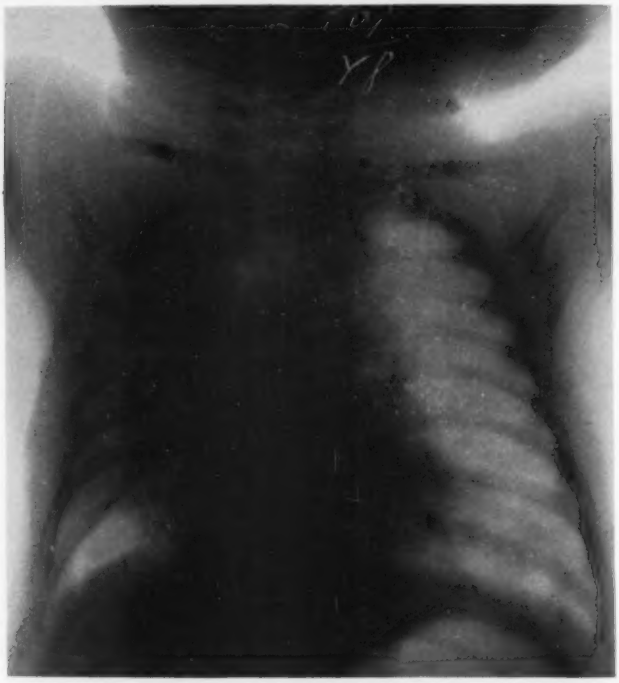

Fig. 4-B. Eight days later. Total atelectasis of the right lung (verified on autopsy 24 hours later). Bronchoscopy not performed.

ETIOLOGY AND PATHOGENESIS OF ACUTE INFECTIOUS ATELECTASIS.

The term "atelectasis" means areas in the lungs where the alveoli contain no air but have undergone pronounced retraction and shrinkage. The prerequisite of this condition is active contraction of muscle tissue in the lungs, which thus distinguishes atelectasis from simple collapse of the lung (Xalabarder, 1949).

Clear-cut atelectasis is merely a stage of brief duration that is followed by vasodilatation with production of a serous, sometimes hemorrhagic, intra-alveolar exudate that may become infected from the bronchi. Atelectasis may be reversible at any of these stages. If it becomes more protracted, however, fibrosis and bronchiectasis may develop in the affected area.

In many cases the atelectasis process is preceded by bronchial obstruction; but it is the general opinion that it may also develop without obstruction, presumably through reflex action combined with hypoventilation (Xalabarder, 1949). Atelectasis due to obstruction occurs most often in infants. For instance, Anspach (1939) states that 80 per cent of such cases of atelectasis are found in children under two years of age, but he does not offer any explanation of this. Van Allen and Soo, on the other hand, discuss this question, pointing out that "collateral respiration" may prevent the development of atelectasis, even though small branches of the bronchi may become obstructed. In infants, however, the lungs contain relatively far more interstitial tissue than the lungs of older children and adults, resulting in a lesser possibility of "collateral respiration." This perhaps may explain the greater frequency of atelectasis in infants; furthermore, the risk of bronchial obstruction is also greater in infants, because of the small dimensions of the bronchi and the inability of infants to cough and expectorate.

SYMPTOMATOLOGY OF ACUTE INFECTIOUS ATELECTASIS CLINICAL.

The predominant feature in these children is severe dyspnea with pronounced retractions of the epigastrium and jugulum but no stridor. The chest is markedly distended, being almost fixed in the position of maximal inspiration, so that in spite of the great efforts of the thoracic wall, the breathing becomes very shallow. As a rule in such cases no rales are heard on auscultation. It is also a characteristic feature that the dyspnea does not satisfactorily improve with oxygen therapy—in contrast to pneumonia. The color of the face is gray-

ish, associated with a state of shock. Definite cyanosis, therefore, is not a dominant symptom, appearing only occasionally on marked physical exertion, *e.g.*, when the child is crying.

Cough may be entirely absent, but in typical cases a so-called "frustrate" cough—*i.e.*, a dry, hacking, paroxysmal and ineffective cough—is present.

The temperature is often normal or only slightly elevated, as seen in children with severe pneumonia.

On auscultation—as in pneumonia—dullness, impaired respiration and rales may be demonstrated; but a sign of differential diagnostic significance is retraction of the affected side of the chest with displacement of the heart to the affected side, even though, as a rule, this may be ascertained only when the atelectasis involves the right lung and with diminished excursions of the chest wall and, sometimes the diaphragm, kept at a high level.

Bronchoscopy shows a very abundant amount of mucopurulent secretion in the bronchi.

ROENTGENOLOGICAL.

The X-ray picture is characterized by a shadow produced by the retracted atelectatic area, with a sharp convex border; the trachea and the heart are displaced to the affected side; the interlobar fissures are displaced, and the intercostal spaces diminished, sometimes with elevation of the diaphragm.

As these patients were markedly exhausted, the X-ray examination had to be performed in the ward. At first the Roentgenograms were failures, because the very rapid respiration (80 to 100 per minute) required a rather short time of exposure, and this was not practicable with the portable X-ray apparatus at our disposal. This difficulty was overcome, however, by means of a simple trick, *viz.*, by means of a pinprick the child was made to cry, and after a few vigorous screams the breathing stopped long enough for adequate Roentgenography.

DISCUSSION.

The clinical picture just described as characteristic of acute infectious atelectasis differs from that of the typical acute fulminating laryngotracheobronchitis primarily in the absence of any obstruction of the larynx, *i.e.*, dyspnea is the main symptom. On the other hand, it is well known that the occurrence of atelectasis may also play an important rôle in laryngotracheobronchitis—first described by Brennemann, Clifton, Frank and Holinger (1938) in America, and later by Boysen and Boysen (1942) and Arnesen, Bjerkelund and Boe (1947) in Norway, and by Söderling (1947) in Sweden. These authors also mention that in some cases of fulminating laryngotracheobronchitis the laryngeal obstruction may not be obvious or spontaneously may subside completely. In these cases a clinical picture also develops in which "pulmonary dyspnea" results from obstruction of the deeper air passages and possibly the formation of this atelectasis is the dominating feature; however, this clinical picture is probably a rather uncommon variant of fulminating laryngotracheobronchitis. None of these authors differentiate acute infectious atelectasis without stridor as a nosographic entity *per se*, which the clinician must keep in mind, especially in the diagnosis and treatment of pneumonia.

TREATMENT.

Aspiration of the secretion in the bronchi is the most important part of the treatment in that phase of acute infectious atelectasis where the dyspnea is most pronounced, and the patient is threatened with suffocation. As described in our case histories, the aspiration should be performed in connection with peroral bronchoscopy. This causes minimum upset and is easy to perform within a few minutes with the child in bed. It is tolerated well even by moribund infants. The lifesaving significance of this intervention was emphasized by its immediate effect on the condition of the patients and by the aspiration of the surprisingly large amounts of secretion. It is to be emphasized that bronchoscopy with aspi-

ration should be looked upon as indicated whenever the clinical picture is suggestive of infectious atelectasis, even though the presence of this condition has not been verified Roentgenologically.

Tracheotomy, in our opinion, is contraindicated in children presenting this clinical picture, as no laryngeal obstruction was present and because such an operation would mean an added strain upon the child that might easily prove fatal to these exhausted children.

In contrast, both tracheotomy and aspiration would be indicated in typical cases of fulminating laryngotracheobronchitis—as emphasized by Jackson and Jackson (1936), Gittins (1932), and others who later discussed this disease. From their works it is evident that when tracheotomy is performed the aspiration is most often carried out in a "groping" fashion with a catheter inserted through the tracheal cannula. This technique must be considered as less effective than aspiration through a bronchoscope. This latter method is also advocated by Brennemann and collaborators whenever it is technically practicable, *i.e.*, when the instruments required and an experienced bronchoscopist are available.

In more serious cases of simple pseudocroup, tracheotomy will be sufficient in itself.

Besides bronchoscopy, the treatment of severely ill children also requires intensive therapy against the infection, a sufficient supply of fluid, the re-establishment of the electrolyte balance, and possibly blood transfusion in order to overcome the state of shock unquestionably present.

The combating of the infection should be started at once by treatment with antibiotics, etc., in order to ensure the best possible effect, bearing in mind that the nature and resistance of the pathogenic bacteria in the given case are unknown. Naturally, the aspiration should be carried out under sterile conditions, so that the proper bacteriological examination of the secretion and determination of the bacterial resistance may be carried out and further treatment based rationally on the results.

Our patients received both penicillin and streptomycin by injection and inhalation. The results of the cultures and the demonstration of the varying sensitiveness of the isolated bacteria to the antibiotics employed showed this combined therapy to be justified.

CONCLUSION.

1. Acute infectious atelectasis is not a rare condition in infants in connection with bronchitis and pneumonia. Autopsy on 178 infants who died under the diagnosis of pneumonia, bronchitis or laryngotracheobronchitis revealed atelectasis in 43 per cent of the cases.

2. The clinical picture of the lesion is so typical that the diagnosis can be made with a fairly high degree of probability, even on ordinary physical examination, but its verification requires X-ray examination with a portable apparatus on account of the exhausted condition of these children.

3. The most important therapeutic measure is early bronchoscopy and the aspiration of the secretion in the bronchi, and the immediate employment of antibiotics.

4. It seems rather likely that the relatively high death rate from pneumonia in infants despite treatment with penicillin and sulfa preparations is due to unrecognized and, therefore, untreated infectious atelectasis.

SUMMARY.

The development of acute infectious atelectasis is pointed out as a contributory cause of the strikingly high death rate from pneumonia in infancy. The records of two characteristic cases are reported. A brief review is given of the autopsy records for 178 children who died under the diagnosis of pneumonia, bronchitis or laryngotracheobronchitis, showing the presence of atelectasis in 43 per cent. The diagnosis and treatment are discussed. It is emphasized that administra-

tion of sulfa preparations and antibiotics is not sufficient, and that repeated bronchoscopy with aspiration is required in order to avoid suffocation in the most critical phase.

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GRANULOMATOUS ULCER OF THE FACE. A REPORT OF A CASE.*

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There have been extensive reviews of the literature and much discussion as to the etiology and cause of the granulomatous ulcers of the face and adjoining facial structures. Hoover[1] reviewed 60 cases of lethal granulomatous lesions involving facial tissues. Other cases have been reported by Levy,[2] Lierle,[3] Isbell[4] and others. Stewart[5] gave an excellent clinical review in his paper. Williams[6] gave a very lengthy discussion as to the possible causes and associated factors that may play an important part in these lesions. Recently he reported a case[7] that has been treated with hormones of the adrenal cortex. The following case is one in which the causative agent was isolated and the lesion was treated with the appropriate agent both locally and systemically.

A CASE REPORT.

A 74-year-old white female was admitted to the Massachusetts Eye and Ear Infirmary on April 27, 1950, with the complaint of swelling and redness around the lower lid of the right eye. She first noticed swelling on the right side of her nose about Jan. 1, 1950. The swelling and redness gradually increased and involved the right nasal bridge, the right infraorbital region and the cheek. There was no pain, no nasal discharge, no epistaxis, and no previous sinus disease except for a heavy feeling in the right antral region.

Examination revealed an indurated, swollen area over the entire right superior maxilla with marked tenderness over the right glabella region, ascending process of the right maxilla and lacrimal fossa. The infraorbital rim was intact. The buccogingival recess over the canine fossa was full and very tender to palpation. The rest of the physical examination was negative.

The past history revealed that the patient was in excellent health except for mild diabetes which was controlled by diet alone.

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Laboratory data on the first admission was essentially negative except that the X-rays showed some thinning of the right nasal bone and many abscessed teeth. The leucocyte count was 8,700, 59 per cent polymorphonuclear leucocytes.

On admission it was felt that this was possibly a malignant granuloma, but with the finding of many abscessed teeth, it was felt that these should be removed as a possible source of infection. The patient was put on a diabetic diet, 200,000 units of aqueous procaine penicillin and hot compresses to the area of involvement. The abscessed teeth were removed. While the patient was under the above treatment, a large slough developed in the area of the previous swelling. The bone underlying this area looked normal. It was felt that the patient could be followed satisfactorily as an outpatient and she was discharged, to be given daily injections of 300,000 units of procaine penicillin.

While being treated as an out-patient, it was noted that the area of ulceration was extending. A biopsy was taken and the report was returned as chronic inflammation. The patient was again admitted to the hospital and at this time the area of ulceration measured approximately 7 cm. in length by 1.5 cm. in width. There was undermining of the skin edges for approximately 4 mm. The base of the ulcer was covered with a small quantity of granulation tissue, and there was redness and induration extending beyond the ulcerated area (see Fig. 1). The lesion extended from the infraorbital region of the right eye, along

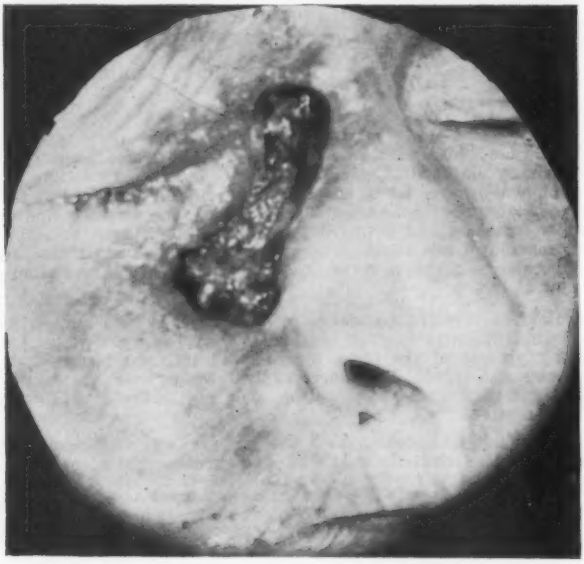

Fig. 1. On admission to the hospital.

the ascending process of the right maxilla to the right nasal bone. It was approximately 3 mm. medial to the inner canthus of the eye.

The laboratory data on this admission revealed a leucocyte count of 8,700 with 67 per cent being polymorphonuclear. Blood sugar was 148 mgm. per 100 cc. of blood. The blood Hinton was negative. Cultures taken on admission and daily thereafter were planted on blood agar plates and meat broth tubes and were placed both aerobically and anaero-

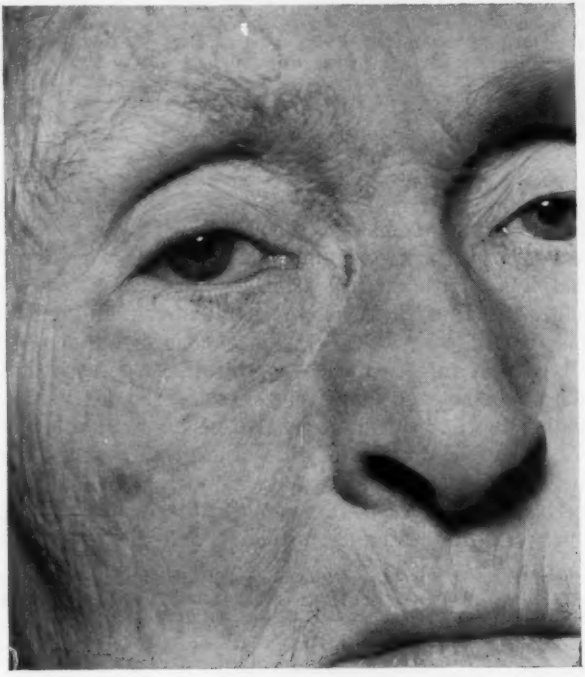

Fig. 2. Upon discharge from the hospital.

bically. The initial culture revealed coagulase positive hemolytic staphylococcus auereus. This organism was found to be resistant to penicillin at 8 units per cc. and very sensitive to aureomycin and bacitracin. Following the report of the cultures and the sensitivity tests, the following plan of treatment was instituted:

1. Cleansing of the ulcer with hydrogen peroxide to help remove all pus and debris. Thorough irrigation with saline to remove all traces of the peroxide and then 10 cc. of bacitracin containing 500 units of bacitracin per cc.

2. Sterile dressing with bacitracin ointment containing 500 units of bacitracin per gm.

3. Aureomycin 3 gm. daily and streptomycin 2 gm. daily.

Following this plan of treatment, on the fifth hospital day, granulations began to appear at the inferior lateral portion. After the sixth hospital day, all cultures were reported as "no growth." By the eleventh hospital day the epithelium had begun to grow from the nasal margin. On the fifteenth hospital day the streptomycin and the aureomycin were discontinued, but the local dressings were applied twice daily. The epithelium gradually closed over the area of the ulcer except for a small fistula that has remained in the region of the lacrimal sac. The patient was discharged from the hospital on the thirty-sixth hospital day. During her stay in the hospital, all urinalyses were negative for sugar. The patient has been followed at weekly intervals in the out-patient clinic and there has been complete healing of the area with minimal scar contraction. Repeated X-rays of the nasal sinuses fail to reveal any changes.

DISCUSSION.

A case of granulomatous ulcer of the face has been presented. The causative organism in this case was felt to be the staphylococcus aureus. This organism was found to be penicillin resistant, but senstive to aureomycin and bacitracin. It was treated with local dressings and oral aureomycin. It is felt that in infection such as this that repeated cultures should be taken and sensitivity results should be done to determine the appropriate agent to be used. In the present day of the great number of chemotherapeutic and antibiotic agents, along with the increasing number of organisms that are becoming resistant to these agents, there must be cooperation between the attending physician and the bacteriologist.

I acknowledge my indebtedness to Miss Anita Mangiaracine, bacteriologist at the Massachusetts Eye and Ear Infirmary, for her valuable bacteriological studies and suggestions as to treatment.

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A CASE OF CHOLESTEATOMA OF THE MIDDLE EAR IN A FIVE-MONTH-OLD INFANT.*

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Although the acute surgical mastoid has become a thing of the past, the problem of the cholesteatoma of the middle ear still remains to torment the otologist. The etiology, pathology and physiology, as explained by Wittmaak,[12] have been generally accepted by the majority of practicing otolaryngologists; however, periodically there appears in the literature a dissenting view.[1,8,9,10] The problem resolves itself to the basic premise: which comes first, the cholesteatoma or the chronic otitis media and mastoiditis? Some men have gone so far as to diagnose every case of chronic otitis media as a type of cholesteatoma.[8]

The rarity of this condition in infancy and childhood and the finding of a cholesteatoma only in the middle ear that has "run" for years make the hypothesis of the infectious etiology of cholesteatoma very plausible;[2,4-6] however, the occasional occurrence of cholesteatoma in an ear that has discharged for only a short period of time and the difficulty in explaining marginal perforations give strength to that group who believe the cholesteatoma to be the primary condition.[10]

When one reviews the history of the early description of cholesteatoma, he is surprised that the pathology and etiology are based on rather casual observations.[11] Wittmaak[12] considered the process of cholesteatoma formation as an attempted repair by nature. The squamous lining of the external canal grows into the middle ear to replace the

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degenerating mucous membrane. The formation of cholesteatoma will occur only if there is an inadequate outlet for drainage of the exfoliating material. Thus the attic and antrum, because they have a relatively small outlet, are ideally located for this formation. His views have been generally accepted, although he carried on no conclusive experiments to prove them.

Tumarkin,[3] on the other hand, feels that the cholesteatoma arises from the normal inflammatory reaction of the pavement epithelium of the epitympanum, retrotympanum and pretympanum. He believes that the pavement epithelium, which normally lines these areas, reacts to infection (and possibly other irritants) by proliferation and the throwing off of paper-like squamas. If the epitympanum, retrotympanum and pretympanum react as described, the above explanation of cholesteatoma becomes very plausible.

Kopetsky[4] has made a very practical division of cholesteatoma into the true and pseudo types. The pseudocholesteatoma is further divided into the primary and secondary. The primary type is associated with a dry ear and is believed to be due to retraction of the drum and then perforation; the secondary type is associated with a draining ear. It is generally known that a condition of true cholesteatoma can occur anywhere in the skull and is rarely (except when the temporal bone is involved) a direct concern of the otologist. Perhaps in the future the primary pseudocholesteatoma will be explained on the basis of Tumarkin's theory.

Recently at the Children's Hospital of Michigan a case of cholesteatoma in a five-month-old infant was observed. The case report follows:

Present Illness: D. T., a five-month-old white male child, was apparently perfectly well until one week prior to admission, Feb. 26, 1950, when he developed a cold with cough, running nose and irritability. The mother was not concerned until the morning of admission when she first noticed an asymmetry of the face. Patient remained irritable and the left ear began to drain. The mother denied any previous trouble, any postauricular swelling or any pulling at the ears. The child had been a normally developing child until onset of the present illness.

Past History: Essentially negative.

Birth and Development: Normal.

Family History: Essentially negative.

Physical Examination: Patient was a five-month-old, well-developed and well-nourished white male child. He was irritable but was in no acute distress and appeared only moderately ill. Hydration was good. Fontanelle was patent but not full. *Eyes:* Left eyelid was paralyzed, and patient could not close the eye. Right eye was normal. Pupils normal. *Ears:* Right drum was mildly injected. In the left external canal some yellow, creamy discharge was seen which obscured the drum. There was questionable mastoid tenderness on the left. The left-sided complete facial paralysis was the only other finding of importance.

Impression: Left acute otitis media and purulent mastoiditis with left facial paralysis.

Laboratory Data: Spinal tap was negative. Blood count showed marked leucocytosis with 29,500 white blood cells of which 51 per cent were neutrophiles. Culture of ear discharge revealed D. pneumonia.

Hospital Course: On the day of admission the left canal was aspirated. Some grayish green tissue was obtained, along with the purulent discharge and was sent to the pathological laboratory. The left eardrum was then visualized for the first time. There was seen a posterior inferior perforation extending superiorly to the membrana flaccida. The malleus

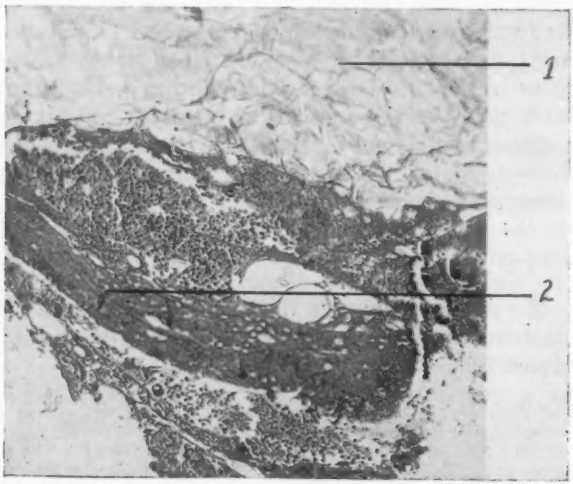

Fig. 1. Cross-section of tissue removed from external ear canal. ×150. (1) Epithelial detritus and keratin sheets in cyst contents. (2) Epithelial wall lining destroyed and desquamated; replaced by connective tissue and leucocytes.

appeared to be intact. The anterior drum was injected. Through the perforation some of this grayish material was seen in the middle ear. The external canal appeared normal. The following day similar material was again present, again removed and sent to pathology. The drum had the same appearance. On March 8, 1950, a pathological report of cholesteatoma was made on this tissue (see Fig. 1). The facial paralysis persisted. X-rays were taken but were of no diagnostic significance.[7] On March 8, 1950, patient was taken to the operating room and under local anesthesia a left cortical (simple) mastoidectomy was done. The bone was sugary in consistency, and in the antrum, in addition to much pus, more of this grayish green tissue was found, sent to the pathological laboratory and again a diagnosis of cholesteatoma was made (see Fig. 2). Drains were placed in the mastoid cavity and in the external ear canal. The incision was not closed. The patient left the operating room in fairly good condition. The postoperative course was complicated by a very severe diarrhea, of questionable etiology, but patient finally recovered. Postoperatively he received penicillin and streptomycin and, at the height of his diarrhea, he was started on aureomycin. An adhesive tape splint was used on the left side of the face, and patient was given physiotherapy. On the twenty-seventh postoperative day he was discharged. The postauricular incision had hea'ed; the left eardrum was intact; and there was questionable improvement of the left facial paralysis.

He was followed in the out-patient department, and his mother was to give him physiotherapy at home. On May 17, 1950, it was noted that the facial paralysis was no longer present; face moved symmetrically. The eardrums were examined and both appeared normal.

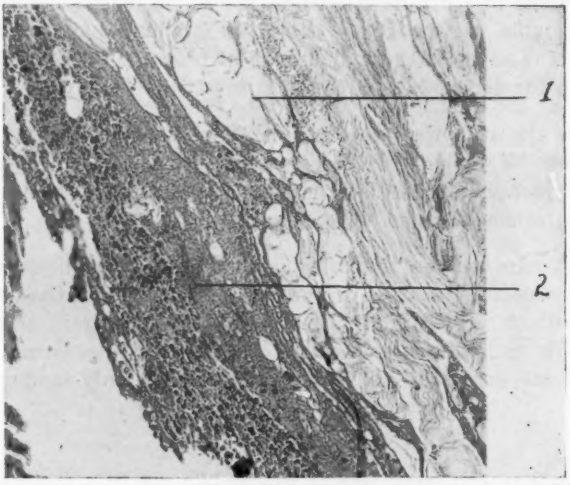

Fig. 2. Cross-section of tissue removed from mastoid cavity. ×150. (1) Epithelial detritus and keratin sheets in cyst contents. (2) Epithelial wall lining partially destroyed and replaced by connective tissue and leucocytes.

On June 14, 1950, patient returned for a routine postoperative visit. At that time the mother stated that the child had developed a cold with cough and nasal discharge one week previously. Two days prior to the visit, there was noticed a purulent discharge coming from the right ear and one day previously it was noticed that the right side of the face was paralyzed. On aspirating the right canal, there was seen a central perforation with a pulsating, purulent discharge. Ear culture revealed hemolytic staphylococcus aureus. There was questionable right mastoid tenderness and a complete right-sided facial paralysis. The left ear was normal. The left face was normal. The patient was admitted, and on June 17, 1950, a right cortical (simple) mastoidectomy was done. The findings were those of an acute mastoiditis. No cholesteatomatous material was found. Patient was placed on penicillin and sulfadiazine. The postoperative course was uneventful, and on July 1, 1950 (20 days postoperatively), patient was discharged home. At that time his postauricular incision had healed, both drums were intact, and the facial paralysis on the right was minimal.

On July 28, 1950, patient was seen in the out-patient department. At that time there was no facial paralysis, the right and left drums were normal, and patient was doing very well.

COMMENTS.

This case, in itself, cannot solve the problem of cholesteatoma of the middle ear; however, the occurrence of a cholesteatoma in a five-month-old child without previously known ear trouble is worthy of note. We realize that Hemsath[3] reports changes in the middle ears of newborns and embryos, due to foreign body reaction and infection.

It is the author's opinion that the work of Tumarkin[5] concerning the etiology of cholesteatoma deserves careful study. The evidence he presents is certainly logical, although his case presentations are not too conclusive.

This case proves that one cannot rule out cholesteatoma because of the duration of the aural discharge or the age of the patient. The occurrence of the facial paralysis, first on one side and then the other, is certainly an interesting phenomenon—especially since the paralysis was only temporary.

SUMMARY.

A case of cholesteatoma of the middle ear in a five-month-old infant is presented. The problem of etiology and pathology is discussed.

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CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The next meeting of the Central Illinois Society of Ophthalmology and Otolaryngology will be held at the Dunlap Hotel in Jacksonville, Ill., on Sept. 21, 22 and 23. The guest speakers will be Dr. Theo. E. Walsh, of St. Louis; Dr. James H. Allen, of New Orleans, and Mr. Henry C. Black, Professional Management, of Detroit.

ILEME CONGRES FRANCAIS D'OTO-RHINO-LARYNGOLOGIE.

The Congrès Francais d'Oto-Rhino-Laryngologie will be held in the Grand Amphithéatre of the Faculté of Médicine, Paris, Oct. 22, 23, 24 and 25, 1951, under the direction of the presiding Dr. M. Bouchet, of Paris, and Honorary President de M. le Prof. Léon Binet, member of the Institute.

For further information, address Dr. H. Flurin, Secrétary Général — 19 Avenue MacMahon, 17e, or Dr. H. Guillon, Assistant Sécretary Général, 6 Avenue MacMahon, Paris, 17e.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

August 1, 1951.

Audicon Models 400 and 415.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Audivox Model Super 67.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1.
Manufacturer: The Dahlberg Co., 2730 W. Lake St., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H Maico; Maico Model J.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303; Micronic Star Model. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model 45.
Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-1 (Whisperwate).
Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model J (Tiny-Myte); Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).
Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.
Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Rochester Model R-1; Rochester Model R-2.
Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)
Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.
(See Micronic.)

Silvertone Model 103BM.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.
Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model J-92.
Manufacturer: Sears-Roebuck Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.
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Silvertone Model P-15.
Manufacturer: W. E. Johnston Mfg. Co., 708W. 40th St., Minneapolis, Minn.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube Model No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., successor to Western Electric Hearing Aid Division, 259 W. 14th St., New York 11, N. Y.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

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Precision Table Hearing Aid.
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Sonotone Professional Table Set Model 50.
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All of the Accepted hearing devices employ vacuum tubes.

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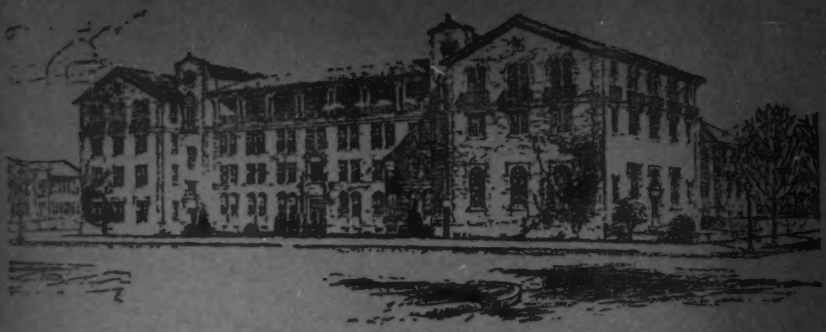

Central Institute for the Deaf

NATIONAL RESIDENTIAL AND DAY SCHOOL FOR THE DEAF AND DEFECTIVES IN SPEECH

Approved by Advisory Council of Foremost Ear Specialists and Educators

New fire-proof buildings beautifully located opposite Forest Park. Modern Dormitories and Equipment. Best home environments. Pupils constantly in care of teachers or experienced supervisors.

ORAL SCHOOL FOR DEAF CHILDREN

C. I. D. offers all advantages of exclusively Speech Training and expert medical supervision for both Resident and Day Pupils.
Nursery School (2 years of age) through the Elementary Grades.

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Private and Class Instruction for children with normal hearing and delayed speech or defective speech.
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